The Next Annual Meeting of the Massachusetts Medical Society Will Be Held in Springfield, June 8, 9, 10, 1936, at the Hotel Kimball and the Springfield Auditorium

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## CONTENTS

Total Thyroidectomy for Heart Disease. *Richard J. Clark; James H. Means* and *Howard B. Sprague* ..... 277

NEW ENGLAND SURGICAL SOCIETY

The Contribution of the Community Hospital to Better Medical Service. *Peer P. Johnson* ..... 295

Emotion and Diarrhea. *Albert J. Sullivan* ..... 299

NEW HAMPSHIRE MEDICAL SOCIETY

Treatment of Burns. *Grover C. Penberthy* ..... 306

The Heart in Rheumatic Fever. *Clifford L. Derick* ..... 310

Miscellany ..... 316

CASE RECORDS OF THE MASSACHUSETTS GENERAL HOSPITAL

Case 22071—*Frederick T. Lord, Aubrey O. Hampton, Donald S. King* and *Tracy B. Mallory.*
Case 22072—*Chester S. Keefer, Aubrey O. Hampton, Fuller Albright, John H. Talbott* and *Tracy B. Mallory* ..... 317

EDITORIALS

"Drug to End Pain in Angina Reported" ..... 326

The Massachusetts Medical Society. Section of Radiology and Physiotherapy ..... 326

The Issue: Why Does Massachusetts Not Protect Its Citizens? ..... 327

This Week's Issue ..... 328

*(Contents continued on next page)*


# LOBAR PNEUMONIA AND SERUM THERAPY

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## CONTENTS

*(Continued)*

THE MASSACHUSETTS MEDICAL SOCIETY
Section of Obstetrics and Gynecology. Analgesia During Labor ..... 328

BOOK REVIEW
The Sexual Relations of Mankind. Paolo Mantegazza ..... 340

CORRESPONDENCE
Cloud Appearances Resembling Pathologic Conditions as Shown in X-Ray Pictures. Wm. Pearce Coues ..... 336
Pseudo Medical Hocus Pocus. William Dameshek ..... 335

MISCELLANY
Appointment of Sir Frederick Hopkins ..... 329
Appointment of Dr. M. J. Rosenau ..... 329
Comparison of Disease Incidence in Connecticut with 1935 and Seven Year Average Month Ending February 1, 1936 ..... 330
Honor to Dr. Karl Bowman ..... 330
Maine News ..... 329
Massachusetts Department of Public Health, January, 1936 ..... 331
Medical Affairs in Connection with the California Pacific International Exposition ..... 294
Morbidity and Economic Problems of Citizens of Boston ..... 332
Prevention of Decay of Teeth ..... 305
Summary of Mortality from Automobile Accidents ..... 335
NOTICES ..... 338
NOTICES OF MEETINGS ..... 338
OBITUARY ..... 337
RECENT DEATH ..... 337
SOCIETY MEETINGS, CONGRESSES AND CONFERENCES ..... 339

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**The next Annual Meeting of the Massachusetts Medical Society will be held in Springfield, June 8, 9, 10, 1936.**

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# TOTAL THYROIDECTOMY FOR HEART DISEASE*

## Experiences With Twenty-One Patients at The Massachusetts General Hospital

BY RICHARD J. CLARK, M.D.,† JAMES H. MEANS, M.D.,† AND HOWARD B. SPRAGUE, M.D.†

### *I. Introduction.*

WHEN a new form of therapeutics is invented and seems to rest on sound theoretical considerations and is backed up by encouraging results in the hands of the inventors, it is very desirable that it be given adequate and careful trial by other professional groups.

The proposal, by Blumgart, Levine and Berlin in 1933, of total ablation of the normal thyroid for the relief of heart disease seemed to us to rest on a reasonable theoretical basis[1]. We also had the opportunity, very early in the history of this form of treatment, to examine the patients at the Beth Israel Hospital who had been subjected to the operation in question and were very favorably impressed with their seeming improvement.

The theory underlying this new practice was elemental in its simplicity. If the heart has so much work to do that it becomes inadequate, give it less to do and perhaps it will again become adequate. Or in the case of angina pectoris, if the coronaries are not capacious enough to allow the blood the myocardium's metabolism requires to pass with ease, decrease the myocardium's oxygen requirement and the coronaries may then become relatively adequate[2]. In both cases it is an adjustment of demand for function to supply of function, that is to say, functional capacity on the one hand of the heart muscle as a pump, on the other of the coronaries as passageways.

There was also considerable past experience which lent support to these theories. Patients with myxedema have long been known to develop angina pectoris occasionally when given thyroid. Thyrotoxic patients with congestive heart failure have regained cardiac compensation when their thyrotoxicosis has been abolished. One is justified in relating these phenomena to shifts in the level of metabolism.

Previously, detailed studies of the heart in forty-eight myxedema patients had been made at this clinic. We showed that while cardiac enlargement was frequently present, decreasing under thyroid medication, and while electrocardiographic changes were always present, there were no patients with signs or symptoms of congestive failure attributable to the "myxedema heart"[3, 4].

For these various reasons it was decided in 1933 to make a trial of the therapeutic method of Blumgart, Levine and Berlin at the Massachusetts General Hospital. We considered that our approach should be primarily from the viewpoint of clinical rather than laboratory investigation. We wished to determine for ourselves the practicability and usefulness of the method for the treatment of cardiac patients in a large general hospital without unduly elaborate special service set-ups or without special technicians or nurses working on the problem, and without special added expense.

We shall first report our findings and then attempt to evaluate them.

### *II. Patients Studied.*

This report is based on a study of twenty-one patients operated on between July, 1933 and May, 1935. The three patients operated on in 1935 are now dead. The last patient to be operated on who is still surviving, was done in August, 1934 and has been followed for fourteen months. Therefore all patients have been observed for a sufficient period of time to make a reasonable evaluation of results. An etiological summary of the cases is presented in table 1.

TABLE 1

| | |
|---|---|
| 21 Cases. | 15 Ward Patients. 6 Private Patients. |
| 19 Cases of Congestive Failure. | |
| | 12 Rheumatic Heart Disease. |
| | 4 Rheumatic and Hypertensive Heart Disease. |
| | 2 Hypertensive Heart Disease. |
| | 1 Syphilitic Heart Disease. |
| 2 Cases of Angina Pectoris. | |
| | 1 Pure Angina. |
| | 1 Hypertensive Heart Disease with Angina and Failure. |
| 10 Males. | 11 Females. |
| Ages 27 to 67. | Average 42½ years. |

*From the Thyroid and Cardiac Clinics of the Massachusetts General Hospital.

†Clark, Richard J. — Assistant in Medicine, Massachusetts General Hospital. Means, James H.—Professor of Clinical Medicine, Harvard University Medical School. Sprague, Howard B.—Assistant Physician, Massachusetts General Hospital. For records and addresses of authors see "This Week's Issue," page 328.

Obviously this report is chiefly one of results in congestive failure. The paucity of angina cases comes from no prejudice against the procedure for them, but simply from the fact that the angina patients seen here have either had inadequate medical treatment, or have been too seriously sick for operation, or have been given alcohol nerve injections by preference.

This report includes no patient with thyrotoxicosis. In all instances the ablated thyroid gland was normal histologically.

*III. Methods of Procedure.*

Patients seen in the Out Patient Department whom it was felt might benefit from total thyroidectomy have been sent into the medical service for further study. There, after complete workup, they have been seen by members of the cardiac and thyroid clinics, and of the surgical service who have given their opinions as to the feasibility of operation.

On the day of operation, the patients have been transferred to the surgical service. The operations have been performed by one of three surgeons, Dr. Arthur W. Allen, Dr. Edward D. Churchill or Dr. Richard H. Miller. The patients have in some cases been discharged directly from the surgical ward, and in other cases have returned to the medical ward for further convalescence before discharge. In all instances the medical men have followed the patients in the surgical wards, and every effort for coöperation between the two services has been made.

Following discharge from the hospital, patients have been followed at regular intervals by one of us (R. J. C.) in the metabolism laboratory. He has had the constant assistance of various members of the thyroid and cardiac clinics.

Six of the patients reported in this series are private ones operated on in the Baker Memorial or Phillips House. They have been selected and followed in much the same way as the ward patients, except that the follow-ups have been made by their own physicians. We are indebted to Dr. P. D. White, Dr. H. B. Sprague and Dr. John Cass for permission to include their patients in this report.

The evaluation of results presented here has been made following considerable deliberation. It takes into account the opinions of members of the patient's family in some cases, in some cases that of the family physician or the patient's private consultant, but finally it represents the opinion of the writers who have seen these cases through their course. Criticism of selection and evaluation of results have also been checked over, on the basis of case summaries, by members of another hospital clinic carrying on the same work. The agreement as to classification of patients has been surprisingly close.

*IV. Selection of Cases.*

The proper selection of cases for total thyroidectomy presents perhaps the greatest and the most important problem of all. In the early months we tried to be guided largely by the previous short experience of those in the pioneer clinics. That we have made several errors in judgment will be apparent on inspection of the data. We have continued to learn from our own experience and from that of others. On the basis of this experience at the present time we would list the following as cases in which operation is definitely contraindicated.

1. Patients that have not been given the benefit of entirely adequate medical treatment over a sufficient period of time for full evaluation of its results.
2. Patients showing a rapid progression in spite of adequate medical care. A case such as No. 1 (see below) with gross breaks in compensation at yearly intervals may be considered as slowly progressive. Cases such as No. 17 and No. 21 with second breaks in compensation within three months of the first in spite of rest and adequate care are too rapidly progressive. Cases of syphilitic heart disease as No. 7 are per se too rapid in their downward course.
3. Patients with such severe heart disease that they are unable to establish and maintain compensation on digitalis and bed rest. This eliminates cases with persistent ascites or hydrothorax as No. 6.
4. Patients with high grade mitral stenosis or other mechanical obstruction giving rise to high venous pressure sustained after compensation has been restored.
5. Patients with a low preoperative basal metabolism. Generally minus fifteen is considered to be the borderline, but as may be seen in case No. 16, an angina with a good result, the preoperative level was minus seventeen.
6. Patients with chronic pulmonary disease of any type. The possibility of pulmonary infarction or thrombosis in cardiac patients as a cause for rapid failure in spite of adequate treatment has recently been emphasized[5], and when it has been suspected should certainly contraindicate operation. See case No. 21.
7. Patients with severe nephritis — low P.S.P. test or high N.P.N.
8. Patients with malignant or severe hypertension, especially if associated with generalized arteriosclerosis. See cases No. 9 and No. 11.
9. Patients with active rheumatic infection, bacterial endocarditis, or other concomitant infection.

10. Patients with recent coronary thrombosis, within six months.
11. Patients with status angiosus.

Intractable heart disease, incapacitating the patient or making him too uncomfortable in spite of adequately and fully prescribed care, not eliminated by one of the above contraindications, may be considered to constitute an indication for operation.

It has been our experience that the distressing symptoms of precordial ache, paroxysmal dyspnea and palpitation have been the symptoms most readily relieved. The fatigue and weakness of the cardiac rarely seem relieved and perhaps the fatigue is even greater in the hypothyroid state. "I am no longer conscious of my heart's beating," or words to that effect is often said by patients after the operation.

*V. The Operation and Complications.*

The operative technique of total thyroidectomy has been described fully elsewhere[6]. It should be emphasized that this procedure is a distinctly major one, requiring special training and skill, and a precise knowledge of the anatomy of the field. To be successful all thyroid tissue must be ablated and injury of the recurrent laryngeal nerves and accidental removal of parathyroid tissue must be carefully guarded against.

In our series the duration of the operation has varied from one hour up to two hours and forty minutes, with an average time of two hours and ten minutes for completion.

*Anesthesia.* In the first case of the series regular ether anesthesia was used successfully. All subsequent operations were started under local anesthesia (cervical nerve block and local infiltration). In one case supplementary ether and in another supplementary gas was required because of marked restlessness. Considerable judgment and care have proved to be necessary in the selection of the type and amount of preoperative medication, and this must be varied with every case. These patients do not withstand large amounts of sedation and it has been pointed out that it is not wise to have them too drowsy. We have found it advisable to test out the patients for drug idiosyncrasy several days before operation. The average amount of sedation used has been three grains of amytal or a similar drug at bed time, the same repeated two hours before operation with morphia one sixth grain on call to the operating room. Cases No. 8 and No. 12 may be cited as examples of oversedation. In case No. 8 scopolamine was used without previous test dosage and the excitement caused by it, necessitated further morphia and ether, probably contributing to the fatal outcome.

*Operative Deaths.* There have been three operative deaths in this series. Two of these, cases No. 8 and No. 12 may have resulted from oversedation, and in case No. 8 quite likely from the added factor of supplementary ether. The third death, case No. 9, arose from the operation having been done on a patient who was too seriously sick to withstand the procedure and who should not, we now realize, have been selected for this form of treatment. (See below.)

*Nerve Injury.* We have been fortunate in having only one nerve injury, the right recurrent laryngeal being cut in the second patient of the series. It might be added that this was the first total thyroid ablation by the surgeon involved.

*Hypoparathyroidism.* There have been no cases of frank tetany following operation. There have been three persistent cases of hypoparathyroidism (No. 3, No. 5, and No. 19) and two transient cases (No. 1 and No. 13). None have shown more than subjective symptoms and positive Chvostek signs, and all have been readily controlled by calcium given orally.

TABLE 2

| | |
|---|---|
| 21 Operations. | |
| Average Duration | 2 hours 10 minutes |
| Operative Deaths | 3 |
| Nerve Injury | 1 |
| Hypoparathyroidism | 5 cases |
| Transient | 2 |
| Persistent | 3 |

*VI. Postoperative Management and Complications.*

The majority of the patients have left the hospital from two to three weeks after the operation. At the time of discharge the metabolism has usually shown a drop of ten to fifteen points. Instructions are given to lead a quiet chair and bed life for the following two weeks after which time a reëxamination is made at the clinic, and gradually increasing activity is permitted if the metabolism has shown a satisfactory drop. The rapidity of fall in the basal metabolism has been extremely variable. Usually one month after operation the rate has been in the minus twenties, but without the patients showing signs of definite myxedema.

It may be said that there seems to be considerable discrepancy between the patient's appearance and his basal metabolism test. Roughly a level of between minus twenty and minus thirty has seemed to be the optimum range for maintenance, but some have shown definite myxedema signs at minus twenty, while others have maintained a level of minus thirty without marked signs.

In some cases signs and symptoms of myxedema requiring thyroid for the patient's com-

fort have appeared within two months, and in other cases have not appeared for five to eight months. Metabolic rate readings have ranged from minus eighteen to minus thirty-seven at the time thyroid was started. All of the patients surviving a period of six months have required some thyroid, excepting case No. 3 which has shown thyroid regeneration and case No. 6 where over the survival period of two years a rate of minus thirty was maintained without untoward myxedema symptoms. In all instances thyroid has been started on the basis of symptoms such as marked swelling of the eyes with lacrimation, extreme coldness, or undue somnolence, rather than on the basis of the metabolic rate. It has been the aim to keep the metabolism as low as compatible with comfort.

Very small dosage of thyroid has often sufficed. A quarter grain dose of thyroid every other day has frequently been sufficient to bring about a distinct change in the appearance and feeling of these people. In some cases one quarter grain daily has been required, but rarely more.

Digitalis dosage at first has been maintained as before operation, but in a number of the cases tolerance appears to have been decreased with the lowering of the metabolic rate and it has been necessary to cut the regular allowance in half. Several mild cases of digitalis intoxication appeared.

Menorrhagia, thought to be associated with the hypothyroidism, has appeared in two patients of the series, and has required x-ray therapy.

The majority of the males have been impotent following the operation.

In one case, a patient with hypertrophic arthritis and poor peripheral circulation, joint pains were more severe following operation and attacks of intermittent claudication occurred.

In several cases abdominal distention was a troublesome feature, but increase in thyroid usually brought relief from this.

Auricular fibrillation was present in fifteen cases before operation, but in only two did it cease following operation. In one of these, paroxysms of fibrillation have occurred since and have been quite troublesome.

Mental slowing has been present to a certain extent in about half of the cases.

Regular follow-up at one to three month intervals has been necessary. In some cases the patients have leveled off well on fixed rations of thyroid and digitalis after the first few months. In others frequent readjustment of dosage has been found necessary and it has been with some difficulty that the middle course between that of severe cardiac symptoms and troublesome myxedema symptoms has been followed. (See case No. 15.)

*VII. Results.*

The evaluation of results in a given case is most difficult. The inherent uncertainty of prognosis in the cardiac patient must always be kept in mind. Against the benefits in certain cardiac symptoms must be balanced the undesirable effects of myxedema. Again a patient may show decrease in one cardiac symptom, but if consigned to an increased life of uncomfortable invalidism the operation is hardly to be considered worth while.

We have classified the patients in two ways. First we have grouped them under one of the following classifications:

A. No recurrence of signs or symptoms. Activity increased. Excellent result.
B. Symptoms less severe with increased activity. Moderately improved.
C. Symptoms less severe without increased activity. Slightly improved.
D. No improvement. Poor result.

Secondly, we have tried to determine whether all factors considered, the operation was worth while.

Table 3 presents a comprehensive survey of the patients studied with the classification made under "Results" and "Operation Worth While". A Summary of these results in the total group is presented in table 4.

TABLE 4

| Summary of Results | Total Group of 21 Cases | | |
|---|---|---|---|
| A. 1 case | | | |
| B. 3 cases | Worth While | | |
| C. 5 cases | Yes | 5 | 23.8% |
| D. 12 cases | No | 16 | 76.2% |

(1 case of thyroid regeneration)

There are eight cases which in retrospect and which in view of our present knowledge we would not consider suitable for operation for the reasons given below.

*Case No. 2.* Heart disease too severe. Patient developed ascites and hydrothorax on bed rest and digitalis. Done as a last resort.

*Case No. 6.* Heart disease too severe. Patient had a persistent ascites in spite of bed rest and digitalis.

*Case No. 7.* Heart disease too rapidly progressive. Syphilitic heart disease.

*Case No. 9.* Severe hypertension and generalized arteriosclerosis. Patient was too sick for operation.

*Case No. 11.* Heart disease too severe with marked hypertension.

TABLE 3

| Serial No. | Sex | Age | Diagnosis | Date of Operation | Anesthesia | Nerve Injury | Hypo-para-thyroidism | Date of Death | Poorly Selected Cases | Result | Operation Worth While | Notes |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 1. | F. | 35 | RHD and HHD | 7/13/33 | Ether | 0 | SLT 2 wks. | 7/17/35 | — | B | Yes | Died of acute infection. ? Bacterial Endocarditis. |
| 2. | F. | 48 | RHD | 8/8/33 | Local | X | 0 | 11/2/33 | X | D | No | Disease too severe. |
| 3. | F. | 30 | RHD | 8/15/33 | Local | 0 | Mild Perm. | — | — | D | No | Thyroid Regeneration 16 mos. |
| 4. | F. | 34 | RHD | 9/1/33 | Local | 0 | Mild 10 mos. | 1/18/35 | — | C | No | Died of Bronchopneumonia. |
| 5. | M. | 52 | HHD and Angina | 9/27/33 | Local | 0 | 0 | 12/11/33 | — | D | No | Died of Pulmonary Infarction. |
| 6. | F. | 43 | RHD | 11/7/33 | Local | 0 | 0 | 10/25/35 | X | D | No | Too severe. Persistent Ascites. |
| 7. | M. | 50 | SHD | 11/8/33 | Local | 0 | 0 | 11/24/33 | X | D | No | Disease too rapid and severe. |
| 8. | M. | 42 | RHD | 11/8/33 | Local and Ether | 0 | 0 | Op. | — | D | No | Death probably from Oversedation. Ether. |
| 9. | M. | 54 | HHD | 11/9/33 | Local | 0 | 0 | Op. | X | D | No | Too sick for operation. |
| 10. | M. | 60 | HHD | 11/10/33 | Local | 0 | 0 | 6/28/35 | — | C | Yes | Myxedema troublesome. Died of Cerebral Hem. |
| 11. | F. | 47 | RHD and HHD | 11/18/33 | Local and Gas | 0 | 0 | 7/20/35 | X | D | No | Disease too severe. B. P. 250/120. |
| 12. | M. | 33 | RHD | 2/16/34 | Local | 0 | 0 | Op. | — | D | No | Op. death. Oversedation. |
| 13. | F. | 39 | RHD | 2/20/34 | Local | 0 | SLT 1 wk. | — | — | B | Yes | Excellent for 18 mos. Now in failure. |
| 14. | M. | 34 | RHD | 4/4/34 | Local | 0 | 0 | — | — | A | Yes | Continues very well. |
| 15. | F. | 42 | RHD | 5/18/34 | Local | 0 | 0 | — | — | D | No | Living chair and bed life. |
| 16. | M. | 67 | Angina | 6/19/34 | Local | 0 | 0 | — | — | B | Yes | Physician. Now practicing. |
| 17. | F. | 45 | RHD and HHD | 6/20/34 | Local | 0 | 0 | 8/19/35 | X | C | No | Disease too rapid and severe. |
| 18. | M. | 36 | RHD | 8/10/34 | Local | 0 | 0 | — | X | C | No | Living chair and bed life. |
| 19. | F. | 33 | RHD and HHD | 1/18/35 | Local | 0 | Mild Perm. | 3/13/35 | — | D | No | Died of Bronchopneumonia. |
| 20. | M. | 28 | RHD | 1/28/35 | Local | 0 | 0 | 9/18/35 | — | C | No | Died of Acute Pulmonary Edema. |
| 21. | F. | 27 | RHD | 5/13/35 | Local | 0 | 0 | 10/30/35 | X | D | No | Disease too rapid and severe. |

LEGENDS

RHD—Rheumatic Heart Disease
HHD—Hypertensive
SHD—Syphilitic
OP.—Operative Death
SLT—Slight
Perm.—Permanent

*Case No. 17.* Heart disease too rapidly progressive.

*Case No. 18.* Heart disease too severe and too rapidly progressive.

*Case No. 21.* Heart disease too rapidly progressive. Previous pulmonary infarction.

Assuming that in any future operations we would not select patients of the above type, it seems worth while to see what their elimination from the present series does to our figures. This calculation is presented in table 5.

TABLE 5

SUMMARY OF RESULTS IN 13 WELL SELECTED CASES

| | | | | |
|---|---|---|---|---|
| Group A. | 1 case | | | |
| Group B. | 3 cases | Worth While | | |
| Group C. | 3 cases | Yes | 5 | 38.5% |
| Group D. | 6 cases | No | 8 | 61.5% |

Assuming that with a larger series and greater experience, perfection in operative technique and handling might be attained, it may be fair to examine the results eliminating from the above the one case of failure of total thyroid ablation (No. 3), and the two cases of operative death which probably arose from unwise sedation (No. 8 and No. 12).

TABLE 6

SUMMARY OF RESULTS IN 10 WELL SELECTED AND WELL MANAGED CASES

| | | | | |
|---|---|---|---|---|
| Group A. | 1 case | | | |
| Group B. | 3 cases | Worth While | | |
| Group C. | 3 cases | Yes | 5 | 50% |
| Group D. | 3 cases | No | 5 | 50% |

The present series is not of sufficient size to make significant comparison of results in special types of heart disease or in specific valve lesions. Regarding angina pectoris, we have only two patients, one doing well and one doing poorly. In the two cases of straight hypertensive heart disease with failure, one was poorly selected and did poorly; the other was classed as a C result but seemed to be worth while. Of the four rheumatics complicated by hypertension, two were poorly chosen, one was a B result and definitely worth while, and one was a D result and not worth while. Of the twelve straight rheumatics, four were poorly chosen, one was an A result, one was a B result, two were C results and four were D results; two of this group were considered worth while.

## *VIII. Case Summaries.*

For the sake of brevity in a rather lengthy report, details of past history, family history, and laboratory findings are not included in the following summaries where not germane to the subject in question. In all cases N.P.N.'s and renal function were determined to be normal preoperatively. In no case was evidence of active infection such as elevated white count or sedimentation rate present. All cases were checked with chest plates and failed to show any pulmonary disease or unusual mediastinal shadows. There were no cases with any concomitant chronic disease not mentioned. In no case did the pathological examination show evidence of thyroid hyperplasia. When thyroid dosage is mentioned, U. S. P. Thyroid (Armour's) is understood in each case.

CASE 1. H. C. No. 329747. F. Aged thirty-five. Native. Housewife.

*Diagnoses:* Rheumatic heart disease, mitral stenosis and regurgitation, aortic regurgitation, auricular fibrillation, hypertension, congestive failure.

*History:* The patient entered the medical service June 7, 1933. Eleven years previously she was told that she had a heart murmur, but up to that time there had been no history of rheumatic infection or heart trouble. Six years before she had been in bed for four months with migratory joint pains, fever, and "water about the heart". Since that time there had been marked fatigue, dyspnea on exertion, orthopnea requiring four to five pillows, palpitation, localized precordial pains, and a constant cough. During the four winters previous to entry, she had suffered breaks in compensation with painful liver, nausea and gross edema, each attack becoming progressively more severe. During the winter prior to entry she was in bed for four months, and for the following three months, up to the time of entry, was restricted to a bed and chair life without any activity. She was having cardiac asthma two to three nights a week. She had received careful medical care with full digitalization and maintenance.

*Physical Examination:* The patient showed moderate cyanosis. The heart was distinctly enlarged to the left, grossly irregular in action, and showed apical systolic and middiastolic murmurs, also a basal diastolic murmur to the left of the sternum. The blood pressure was 170/90. The lungs were clear. The liver was enlarged and tender. The ankles showed slight edema.

*B. M. R.* level was zero.

*Rationale for Operation:* This patient had been suffering from mild, progressive heart failure for six years and had been totally incapacitated for seven months on medical treatment without prospect for improvement. It was hoped that operation might bring increased comfort and activity.

*Operation:* July 13, 1933. Anesthesia: Ether. Complications: Positive Chvostek and Trousseau were present for two weeks, but controlled by calcium given orally; operative recovery was otherwise normal.

*Postoperative Course:* For practically two years following operation the patient was very much improved. There were no gross breaks in compensation. There was but little dyspnea on exertion, patient slept on one or two pillows, there was no edema, no cardiac asthma, and almost no precordial pain. Palpitation was noted only on going upstairs. She was able to help with the housework and get the night meal for the family. Her B. M. R. after the first month ranged about minus twenty. After the sixth month thyroid was given in amounts varying from one quarter to one eighth grain daily, because of increasing fatigue, coldness and lacrima-

tion. The patient was able to concentrate less well than previously, found difficulty in mathematical calculations and to a slight extent with her memory. During February, March and April 1935 she had severe menorrhagia, requiring hospitalization. No evidence of pelvic tumor could be found and x-ray treatment was given with cessation of flow. About the first of July she reëntered the hospital with an unexplained fever, a palpable spleen and liver, and a few petechial spots. Blood cultures were negative. July 17 she died with the questionable diagnosis of subacute bacterial endocarditis. No autopsy was obtained.

*Result:* This patient represents a very good result with a definitely improved cardiac state up to the time of her death from acute infection two years after operation. From her mild myxedema she had some slight mental impairment and the probable complication of menorrhagia, in spite of which the operation was very much worth while to her.

*Comment:* This proves to have been a well-selected case. While the patient had severe heart disease it was slowly progressive over a period of a number of years, and signs of failure were well controlled by digitalis and diuretics.

Case 2. D. P. No. 306061. F. Aged forty-eight. Italian. Housewife.

*Diagnoses:* Rheumatic heart disease, mitral stenosis and regurgitation, aortic stenosis and regurgitation, auricular fibrillation, congestive failure.

*History:* This patient entered the hospital June 23, 1933 for the second time with congestive heart failure. There was no past history of chorea or rheumatism. For twenty-one years she had noted weakness, dyspnea and mild edema of the ankles off and on. For four years palpitation, cough, epigastric pain and precordial distress had been troublesome. Six months previously she was admitted to the hospital for gross congestive failure with abdominal and chest fluid, which was relieved by digitalis and salyrgan. Following discharge in spite of maintained digitalization and bed rest, she was required to lead a chair and bed existence and again developed gross signs of failure.

*Physical Examination:* There was marked orthopnea and cyanosis. The heart was grossly enlarged to the left, fibrillating and showed mitral and aortic systolic and diastolic murmurs. The blood pressure was 100/80. There was a right hydrothorax extending to the angle of the scapula. The liver was felt six cm. below the costal margin. The abdomen showed a fluid wave and shifting dulness. The sacrum and legs were edematous.

*B. M. R.* level was plus one.

*Rationale for Operation:* This patient's optimal state appeared to be one of congestive failure and the outlook for improvement appeared hopeless. It was felt that it would be fair to offer her the possible advantage of total thyroidectomy. On salyrgan the patient lost fifteen pounds of edema fluid and operation was determined on.

*Operation:* August 8, 1933. Anesthesia: Local. Complications: The right recurrent laryngeal nerve was cut resulting in paralysis of the right cord. There were no signs of tetany.

*Postoperative Course:* The patient was discharged August 23, 1933. During September and October she seemed slightly improved, having less dyspnea and palpitation, and very little edema. However, she continued to lead a chair and bed life. November 1, 1933 the B. M. R. was minus twenty-six. December 14 the patient reëntered the hospital because of increasing ascites, edema and orthopnea of six weeks' duration. The following morning she died.

*Result:* For two months after operation there appeared to be very slight improvement, but it would be difficult to say that this resulted from operation rather than from the prolonged absolute bed rest and diuretics under observation. In spite of the operation, gross failure shortly set in again and proved fatal.

*Comment:* This was one of the first cases to be done and at that time we did not appreciate the fact as we do now that cases having such low cardiac reserve as to develop ascites or hydrothorax on rest and digitalis are too hopeless to be benefited by operation. This patient should never have been operated on.

Case 3. H. P. No. 330660. F. Aged thirty. Italian. Housewife.

*Diagnoses:* Rheumatic heart disease, mitral stenosis and regurgitation, auricular fibrillation, congestive failure.

*History:* This patient entered the hospital July 20, 1933. Six years before she had been in bed three months with rheumatic fever and was then told that she had a bad heart. Four years before there was a recurrence of rheumatism followed by onset of progressive dyspnea and palpitation. Eight months before entry, edema of legs set in and became massive. For two months the patient was confined to her bed. For one week there had been orthopnea requiring the patient to sit up straight. She had been on digitalis for several months.

*Physical Examination:* There was marked cyanosis. Neck veins were distended with the patient sitting up straight. The heart was grossly enlarged showing apical systolic and mid-diastolic murmurs; rhythm was grossly irregular with an apical rate of one hundred and sixty and a pulse of ninety. There were moist râles at both lung bases and the liver was felt six cm. below the costal margin and distinctly tender. There was a small amount of abdominal fluid. The legs and sacrum showed gross pitting edema. Salyrgan and Southey tubes were required for the elimination of excess fluid.

*B. M. R.* Unfortunately only one rate was obtained and this on the morning of operation after sedation the previous night. It was minus sixteen. (Subsequent events suggest true rate was higher.)

*Rationale for Operation:* It was considered that with conditions as they were, the prognosis in this case was very poor and little could be offered other than the possible benefit from total thyroidectomy.

*Operation:* August 15, 1933. Anesthesia: Local. Complications: Positive Chvostek and Trousseau appeared the day following operation and calcium by mouth has been required since. There was no frank tetany.

*Postoperative Course:* Three weeks after operation the B. M. R. was minus twenty-one. Pulse had become regular. The patient was up and about the ward with very much less dyspnea and palpitation, and there was no edema. For about five months there seemed to be some improvement with less dyspnea, palpitation and orthopnea. Then there was a gradual recurrence of edema and swelling of the abdomen. July 1, 1934 she reëntered the hospital in gross congestive failure with pitting edema and ascites requiring paracentesis. Her B. M. R. at that time was minus twenty-nine although there were no signs or symptoms of myxedema. Following this

there was another period of improvement. In October, 1934 she appeared to be very well, having few symptoms and being able to walk a block. However, November 14, 1934 she again was forced to enter the hospital in gross congestive failure. Her B. M. R. was then minus twenty and still no signs of myxedema other than very slight dryness of the skin.

In December, 1934, after leaving the hospital, there was but slight improvement and the patient was confined largely to a chair and bed life. The B. M. R. December 18, 1934 was plus four and following that time has remained above minus five in spite of no thyroid medication.

In April, 1935, conditions improved considerably with a decrease in dyspnea and palpitation. The patient was enabled to be up and around doing some of the housework which she had not been able to do since the operation. Up to the present, October, 1935, she is still maintaining this improved state, although there has recently been some increase in dyspnea. She shows no signs of congestive failure. With this her B. M. R. has remained minus four and she has shown no sign of myxedema, off thyroid.

*Result:* For about five months this patient appeared to be having some benefit from the operation. Following that for sixteen months she was no better than before operation. About sixteen months after operation she showed definite evidence of thyroid regeneration indicating a failure of complete ablation. The striking factor in this case is the marked improvement from the twenty-first month on, with a normal B. M. R. It does not seem that any worth while benefit can be attributed to the operation, and on the other hand a permanent hypoparathyroidism was induced.

*Comment:* This case perhaps demonstrates more strikingly than any other the inherent vagaries of congestive failure and the difficulties of evaluation of these cases. Had she maintained a state of myxedema during the past seven months we should have called her a very good late result.

CASE 4. R. N. No. 323164. F. Aged thirty-four. Italian. Housewife.

*Diagnoses:* Rheumatic heart disease, mitral stenosis and regurgitation, auricular fibrillation, congestive failure, chronic cholecystitis and cholelithiasis.

*History:* This patient entered the hospital for the third time on August 18, 1933. Nine years previous to this she was in bed for five months with acute rheumatic fever. Three years before she first noted dyspnea and palpitation which has continued and become progressive until marked on the slightest exertion. For two years she had been troubled by localized precordial aches, coming both when quiet and on exertion. One year before, August, 1932, she entered the hospital because of increasing dyspnea, palpitation, orthopnea, right upper quadrant pain, nausea and vomiting of a few days' duration. The liver was moderately engorged, there were a few râles at the lung bases but no edema. While in the house she showed paroxysms of fibrillation. A Graham Test was positive and revealed stones in the gallbladder. She was digitalized and sent home improved. Again in February, 1933, she reëntered the house with a story as before. On this occasion she showed a fixed fibrillation and moderate edema of the ankles. With salyrgan and bed rest she improved slightly. Following discharge this time she was confined to a chair and bed life; edema of the ankles returned and became more marked; she slept poorly requiring four pillows; she had three attacks of cardiac asthma; right upper quadrant pain increased and with this nausea and vomiting. She returned for the third time in gross failure.

*Physical Examination:* There was moderate cyanosis. The neck veins were engorged. The heart was grossly enlarged, totally irregular with an apical rate of one hundred and sixteen and a pulse of ninety, and showed apical systolic and diastolic murmurs. Moist râles were present throughout both lungs. The liver was engorged, tender and palpable ten cm. below the costal margin. There was pitting edema of the legs. On complete rest, salyrgan and increased digitalis the edema of the lungs and legs cleared. Total thyroidectomy was decided on.

*B. M. R.* level was plus seven.

*Rationale for Operation:* This patient had been suffering from progressive failure for three years with three gross breaks in compensation. She had been a chair and bed invalid for six months, living an uncomfortable life in spite of adequate medical treatment. With diuretics she had shown adequate reserve to clear her edema. It was felt that total thyroidectomy was the only further measure which might be of value.

*Operation:* September 1, 1933. Anesthesia: Local. Complications: A positive Chvostek and Trousseau developed requiring calcium by mouth for a period of about ten months. No frank tetany or cord injury appeared.

*Postoperative Course:* For the sixteen months following this patient's operation she ran a variable course for better and for worse. For about ten months dyspnea was very much less marked, but following that it increased and became as severe as before operation. Orthopnea was consistently relieved, the patient sleeping well on one to two pillows. Precordial ache and palpitation were considerably relieved. There was no further cardiac asthma. In February she reëntered the hospital with a severe bronchitis and mild signs of congestive failure. In August, 1934, she again entered the hospital because of increasing dyspnea, severe right upper quadrant pain, and mild edema of legs for one month. She again improved on salyrgan. It was felt that a large part of the abdominal pain resulted from gall stones in spite of the chronically congested liver. Whereas for the first ten months she was able to be up and about her apartment, following that she was again restricted to a chair and bed life, having abdominal pain off and on, mild edema of ankles, and râles at the lung bases. Her B. M. R. was maintained about minus eighteen to minus twenty-five. In February, 1934, one quarter grain dosage of thyroid daily was started because of cold, weakness and marked swelling of the eyes. In July, because of a rise in the B. M. R. to minus fourteen, associated with increasing signs of failure, thyroid was reduced to one quarter grain every other day. The patient was quite miserable from extreme cold, puffy eyes, general weakness and sleepiness. She continued with digitalis one and one half and three grains alternate days.

On January 18, 1935 she entered the emergency ward because of a severe cough and fever following a sore throat. The lungs were filled with moist râles and bronchi. The white count was seventeen thousand with eighty-three per cent polys. That night she died apparently of bronchopneumonia. On the day of death her heart showed little change from the previous month.

*Result:* There was some consistent improvement in certain uncomfortable cardiac symptoms, although the patient was distinctly uncomfortable from her myxedema. Activity was slightly increased for ten months but following that it was no greater than

before operation. Possibly her life was prolonged. From the scientific point of view the operation produced definite cardiac benefit. Viewing the situation as a whole we do not consider the procedure to have been worth while for this patient.

CASE 5. F. G. No. 251759. M. Aged fifty-two. Irish. Unemployed.

*Diagnoses:* Hypertensive and coronary heart disease, angina pectoris, auricular fibrillation, congestive heart failure.

*History:* This patient entered the hospital September 6, 1933 for the seventh time because of heart trouble. He had been partially disabled for ten years and almost totally so for five years with dyspnea, palpitation, orthopnea and intermittent edema. For three years he had suffered attacks of crushing precordial pain radiating down the left arm, coming with any exertion, and relieved by nitroglycerine.

*Physical Examination:* These was moderate cyanosis and respiratory distress. The heart was grossly enlarged, sounds of poor quality, and a basal systolic murmur was present; rhythm was totally irregular. The blood pressure was 190/130. There was marked arteriosclerosis. There were râles at the lung bases. The sacrum and legs showed a moderate edema.

*B. M. R.* level was plus twenty-five.

*Rationale for Operation:* Since this patient suffered from congestive failure, which he was able to clear on bed rest, as well as from angina pectoris which was incapacitating, it was felt that total thyroidectomy should offer some benefit especially in view of the elevated metabolism. The operation was done primarily for angina.

*Operation:* September 27, 1933. Anesthesia: Local. Complications: None.

*Postoperative Course:* Following the operation, this patient rested in the hospital for a month and was then discharged to a convalescent home. There, leading a quiet life, the number of attacks increased although no signs of congestive failure appeared. He died suddenly on December 11, 1933, two and a half months after operation of pulmonary thrombosis and infarction, as revealed by autopsy. There was also found to be marked coronary sclerosis and localized areas of fibrosis of the myocardium.

*Result:* In this patient's relatively short postoperative course, we see no improvement in the angina, but on the contrary his anginal attacks became more frequent in number. There was no return of congestive failure in the two and one-half months postoperative course.

*Comment:* The presence of pulmonary thrombosis probably in part explains the unsatisfactory course with this patient.

CASE 6. S. R. No. 287519. F. Aged forty-three. Irish. Housewife.

*Diagnoses:* Rheumatic heart disease, mitral stenosis and regurgitation, ? tricuspid stenosis, auricular fibrillation, congestive failure, ? portal obstruction.

*History:* October 16, 1933, this patient was admitted to the hospital for the twelfth time in six years with congestive heart failure. From the ages of twelve to twenty-five she had frequent attacks of joint pain. For six years prior to this entry there had been dyspnea, palpitation and edema. For three years there had been orthopnea requiring three or four pillows at night, cardiac asthma occurred almost weekly, and activity was limited to walking from one room to another. During this time ascites set in, requiring abdominal taps about every eight days with removal of six to eight quarts of fluid.

*Physical Examination:* There was moderate cyanosis. Neck veins were distended. The heart was grossly enlarged, fibrillating, and showed apical systolic and diastolic murmurs. The blood pressure was 160/90. There was fluid in the left chest and râles at the lung bases. The abdomen was distended with fluid. The liver was palpable twelve cm. below the costal margin. The legs showed a marked pitting edema.

*B. M. R.* level was minus five.

*Rationale for Operation:* Regarding diagnosis, the question was raised whether this case represented simply rheumatic heart disease with a possible cardiac cirrhosis or some other type of portal obstruction giving rise to the marked ascites. It was felt that on theoretical grounds, with the lessened necessary blood flow going with a lowered metabolic rate, the ascites might be relieved. Furthermore it was believed that increased circulatory failure might be prevented. Without previous experience in this type of case, it was decided, with consent of the patient, to proceed with total thyroidectomy on a purely theoretical and experimental basis.

*Operation:* November 7, 1933. Anesthesia: Local. Complications: None.

*Postoperative Course:* For the first nine months postoperatively, the interval between abdominal taps was cut down to about once in two weeks, but following that, they were again required almost weekly. Other signs and symptoms were little if any improved, although asthma did not occur for the first nine months and was of rare occurrence thereafter. Several chest taps in addition to the abdominal taps were required. The B. M. R. remained about minus thirty, but the patient showed no untoward myxedema symptoms. October 25, 1935, two days after her final tap, the patient grew increasingly weak, and died, the exact cause of death being uncertain. No autopsy was obtained.

*Result:* The operation in this case brought about no worth while benefit.

*Comment:* This case illustrates the futility of the procedure in cases of long standing, intractable, severe congestive failure, and in cases with persistent ascites. This patient should not have been operated on.

CASE 7. J. D. B. M. No. 11472. M. Aged fifty. Native. Coal Dealer.

*Diagnoses:* Syphilitic aortitis, aortic regurgitation, congestive failure.

*History:* This patient entered the hospital October 9, 1933. For six months he had presented rapidly progressive signs and symptoms of congestive failure with dyspnea and cough. For two months he had remained in bed with increasing edema in spite of digitalis. At the age of twenty he had had a chancre.

*Physical Examination:* There was moderate cyanosis and engorgement of the neck veins. The heart was grossly enlarged showing a very loud aortic diastolic murmur. Blood pressure was 170/50. There were a few râles at the lung bases. The liver was felt six cm. below the costal margin. There was a moderate amount of abdominal fluid. The sacrum and extremities showed marked edema.

*B. M. R.* not done. Hinton: Strongly positive.

*Rationale for Operation:* Because of failure to improve after a month of hospitalization, it was felt that thyroidectomy as a last resort was justifiable with a hope of decreasing the congestive failure in spite of the ultimate poor prognosis.

*Operation:* November 8, 1934. Anesthesia: Local. Complications: None.

*Postoperative Course:* There was not any apparent benefit or effect from the operation. November 24, 1934, sixteen days later the patient died at home.

*Result:* No benefit from operation.

*Comment:* This again was one of the early cases operated on as a last resort and we have learned that this does not pay. We have also come to feel that the operation is not worth while in cases of syphilitic heart disease because of the rapidly progressive nature of the lesion.

CASE 8. H. K. No. 332819. M. Aged forty-two. Hebrew. Junk collector.

*Diagnoses:* Rheumatic heart disease, mitral stenosis and regurgitation, auricular fibrillation.

*History:* This patient entered the hospital October 20, 1933. For eight years there had been increasing dyspnea on exertion. For four years there had been marked dyspnea, palpitation, orthopnea, and the patient had been unable to work. For two years there had been increasing edema. For three months the patient had led a chair and bed life. Digitalization had been maintained in the Out-Patient Department for eight years.

*Physical Examination:* There was moderate orthopnea, marked cyanosis and the neck veins were distended. The heart was grossly enlarged to the left, fibrillating and showed apical systolic and diastolic murmurs. The blood pressure was 124/80. There were moist râles at the lung bases. The liver was palpable at the umbilicus and tender. There was pitting edema of the sacrum and legs.

*B. M. R.* level was plus five.

On bed rest and diuretics the edema disappeared.

*Rationale for Operation:* In view of the chronically crippled condition of the patient, in spite of adequate medical care, it was felt that thyroidectomy offered the only chance for improvement.

*Operation:* November 8, 1933. Anesthesia: Morphia one sixth grain s.c. at 7.00 A.M. Morphine one sixth grain, atropine one one-hundredth grain and scopolamine one one-hundred and fiftieth grain s.c. at 8.25 A.M. Operation started with novocaine one per cent infiltration at 9.25 A.M. Because of restlessness and excitement, morphine one sixth grain was given at 10.25 A.M. and the remainder of the operation completed under drop ether.

*Postoperative Course:* The patient did well for twelve hours and at that time the blood pressure dropped, temperature rose, lungs became filled with moisture and the patient died.

*Result:* Operative death.

*Comment:* Since the time of operating on this patient we have learned that these cardiacs sustain large amounts of sedation poorly. We have also learned that it is wise to test out reaction to proposed medication in advance of the day of operation. Excitement, possibly from scopolamine, necessitating additional morphine and ether, quite likely led to the termination in this case.

CASE 9. H. K. B. M. No. 11774. Aged fifty-four. Native. Architect.

*Diagnoses:* Hypertensive heart disease, generalized arteriosclerosis, auricular fibrillation.

*History:* This patient was admitted to the hospital November 4, 1933. For five months he had a noted fluttering and pounding of his heart, and for three months there had been dyspnea on any exertion and slight swelling of the feet at night. For ten years there had been a known hypertension. Recently he had been very nervous and was rapidly losing ground.

*Physical Examination:* The heart was markedly enlarged with sounds of poor quality, harsh systolic murmur in the aortic area and fairly loud blowing apical systolic murmur. Blood pressure 220/140. Lungs showed occasional râles at bases. Legs showed a slight pitting edema.

*B. M. R.* was plus twenty-seven. (Patient very nervous and true rate hard to obtain.)

*Rationale for Operation:* This patient was unable to work, nervous, discouraged and having some evidence of disorientation. The operation was done with the hope of delaying his cardiac failure.

*Operation:* November 9, 1933. Anesthesia: Local. Complications: There was Cheyne-Stokes respiration during the operation. The patient did not recover from a state of stupor and died a few hours later.

*Result:* Operative death.

*Autopsy* showed very advanced arteriosclerosis with beading of the cerebral vessels in addition to hypertensive heart disease.

*Comment:* The high degree of hypertension and the marked generalized and cerebral arteriosclerosis would in view of later knowledge be considered contraindications to operation.

CASE 10. S. C. No. 319289. M. Aged sixty. Russian Hebrew. Unemployed.

*Diagnoses:* Hypertensive heart disease, congestive failure, auricular fibrillation.

*History:* This patient entered the hospital for the second time, November 2, 1933. Twenty-five years before he was seen in the Out-Patient Department complaining of palpitation, and at that time showed cardiac enlargement with a total irregularity and pulse deficit. Since then he had been followed, because of increasing dyspnea and palpitation. Fifteen months prior to the present entry he was sent to the medical wards because of increasing heart failure with orthopnea, pulmonary congestion, engorged liver and slight edema. On bed rest, continued digitalis and diuretics he improved. After leaving the house his course was again one of increasing disability, with persistent moderate signs of congestion. Intermittent, localized sharp precordial pains appeared. For six months he had been confined to a chair and bed life, because of severe dyspnea and orthopnea requiring four pillows and palpitation.

*Physical Examination:* The heart was moderately enlarged, no murmurs were heard, the pulmonary second sound was accentuated, and rhythm was grossly irregular. Blood pressure was 210/110. The lungs showed moist râles at the bases. The liver was felt four cm. below the costal margin. There was no edema.

*B. M. R.* level was plus three.

*Rationale for Operation:* This patient was totally incapacitated on full digitalization and showed signs of progressive cardiac failure. It was believed that he could be given greater activity with retardation of terminal failure by the operation.

*Operation:* November 10, 1933. Anesthesia: Local. Complications: None. Uneventful recovery.

*Postoperative Course:* For about fourteen months following operation this patient seemed slightly improved. His activity was increased to the point of walking one quarter to one half mile in a day. Orthopnea disappeared and he slept well on two pillows. There was no palpitation. Dyspnea became distinctly less. There was practically no edema. Precordial aches were almost entirely relieved. On the other hand he felt generally weak and became tired very easily. It was fatigue rather than dyspnea which limited his activity. Because of marked puffiness about the eyes, lacrimation, cold and increasing fatigue, one quarter grain of thyroid daily was started during the second postoperative month and was continued thereafter with the B. M. R. ranging between minus fifteen and minus twenty. Digitalis one and one-half and three grains were continued on alternate days.

During the winter of 1934 and 1935, the patient minded the cold far more than previously. Old hypertrophic arthritis gave increasing pain. Peripheral circulation became very poor, and attacks of intermittent claudication set in. During the spring the patient grew generally somewhat weaker, more dyspneic, and was forced to lead a very quiet life. In June 1935 he had a cerebral hemorrhage from which he never recovered, dying on June 28, 1935.

*Result:* This patient's cardiac status was definitely improved by operation, and impending gross failure was warded off but the side effects of the myxedema were distinctly troublesome and he was all in all a rather miserable patient.

CASE 11. G. K. B. M. No. 11859. F. Aged forty-seven. Native. Housewife.

*Diagnoses:* Rheumatic and hypertensive heart disease, mitral stenosis, auricular fibrillation, congestive failure.

*History:* This patient entered the hospital November 12, 1933. For five years there has been marked dyspnea and palpitation on exertion, and for four years she was able to do little housework because of this. For two years there had also been swelling of the ankles at night and orthopnea. During the previous year there had been five severe attacks of paroxysmal dyspnea, palpitation, cough and frothy sputum, each requiring three to four weeks in bed. The patient had been resistant to digitalis and had required four and one-half grains a day constantly to control the apex rate.

*Physical Examination:* There was moderate respiratory distress and cyanosis. The heart was grossly enlarged, totally irregular and showed an apical diastolic murmur. Blood pressure was 250/120. There were moist râles at the lung bases. The liver was palpable six cm. below the costal margin. There was moderate edema of the legs.

*B. M. R.* level was plus seventeen.

*Rationale for Operation:* This patient was laid up frequently with tachycardia (auricular fibrillation and orthopnea) and was losing ground steadily in spite of adequate medical care.

*Operation:* November 18, 1933. Anesthesia: Local. Complications: None.

*Postoperative Course:* The immediate effect of operation was one of striking benefit. The heart became slow, palpitation disappeared and the patient was able to lie flat without discomfort. There was a great deal of psychic trauma from the operation under a local anesthetic and she was troubled with bad dreams for months afterward on returning home. Dyspnea on exertion, some edema, headache and abdominal distention continued to incapacitate her for any activity, other than walking about her room. Attacks of paroxysmal dyspnea recurred. She again required three to four pillows at night. She was taking three grains of digitalis a day, and one half grain of thyroid every other day was required for myxedema symptoms after the second month.

In March, 1935 (sixteen months after operation) the patient's local doctor felt that she was too myxedematous and not doing well, so raised her thyroid to one grain daily (no B. M. R. was taken), and at the same time increased her digitalis to four and one-half grains daily. Following this some of the puffiness of her face and ankles decreased. Gradually signs and symptoms of failure increased with cardiac asthma occurring almost nightly. Nausea set in and all digitalis was omitted for a week. May 7, 1935 the patient reëntered the hospital in congestive failure. At this time her B. M. R. was plus seven. During an eighteen day rest in the hospital off thyroid her rate fell to a level of about minus fifteen. Digitalization was reëstablished on one and one-half and three grains daily. Symptoms were somewhat improved.

After returning home the patient was again confined largely to a chair and bed life with moderative congestive failure. July 20, 1935 twenty months after operation, she suddenly developed acute pulmonary edema and died within a few hours.

*Result:* There was little if any benefit from the operation in this case and it was certainly not worth while.

*Comment:* This again is a case of very severe heart disease with marked hypertension which should not be selected for operation today.

CASE 12. L. H. No. 263901. M. Aged thirty-three. Hebrew. Unemployed.

*Diagnoses:* Rheumatic heart disease, mitral stenosis and regurgitation, aortic regurgitation, auricular fibrillation, congestive failure.

*History:* This patient was admitted to the medical service January 24, 1934. At the age of thirteen he was in bed for eight months with rheumatic fever. For ten years there had been some increasing dyspnea on exertion. For five years palpitation had become increasingly troublesome. In the previous three years there had been three attacks of congestive failure with cough, orthopnea and liver pain, the last setting in three weeks before entry. Between attacks he had been able to walk only a few blocks without exhaustion.

*Physical Examination:* There was moderate cyanosis and neck veins were pulsating. The heart was grossly enlarged to the left, fibrillating and showed mitral systolic and diastolic murmurs, also an aortic diastolic. The blood pressure was 145/90. There were râles at both lung bases. The liver was palpable and tender eight cm. below the costal margin.

*B. M. R.* level was plus eleven.

*Rationale for Operation:* In view of repeated breaks in compensation without massive edema responding well to bed rest and digitalis, but with an obviously low reserve, it was believed that operation should be of marked benefit in this case.

*Operation:* February 16, 1934. Anesthesia: Luminal three grains at bed time, pentobarbital three grains at 7.00 A.M., pantopon one third grain s.c. at 7.30 A.M., pantopon one third grain s.c. at 8.00 A.M., pantopon one third grain s.c. at 8.30 A.M., operation started at 8.50 A.M. Novocaine one per cent cervical block and infiltration. The thyroid was easily removed in toto. No parathyroids removed. No nerve injury. The patient's condition remained good during the operation.

*Postoperative Course:* The patient's condition re-

mained fairly good up to 4.10 P.M. when he suddenly became cyanotic with irregular, gasping respiration, pulse of one hundred and blood pressure 140/80. He became unconscious; in spite of oxygen therapy, respirations grew weaker and more irregular, blood pressure dropped to 80/40. At 3.35 A.M. the following morning, some sixteen hours following operation he died in apparent respiratory failure.

*Result:* Operative death. No autopsy.

*Comment:* It is possible that a cerebral embolus to the respiratory center may have been the cause of death in this case. Again, as in case 8, we note that a large amount of sedation was given which we have since learned these people tolerate poorly.

CASE 13. E. J. P. P. H. No. 32923. F. Aged thirty-nine. Native. Housewife.

*Diagnoses:* Rheumatic heart disease, mitral stenosis, auricular fibrillation.

*History:* This patient entered the hospital February 12, 1934. Fifteen years before she developed a cough which had continued since, and shortly thereafter dyspnea on exertion set in and grew progressively more marked. During the previous six years palpitation had been severe and constant. For one year there had been frequent localized heartache. Hemoptysis had occurred on four occasions in the previous six years. For three years there had been fulness in the abdomen and on one occasion definite congestive failure requiring ten weeks' rest in bed. Digitalization had been maintained for several years.

*Physical Examination:* The heart showed marked enlargement to the right. There was a long, loud apical mid-diastolic murmur. The rhythm was totally irregular. The lungs were clear. The abdomen was distended with the liver palpable five cm. below the costal margin and slightly tender. There was very slight pitting edema of the lower shins.

*B. M. R.* level was minus nine.

*Rationale for Operation:* Because of marked discomfort and slowly progressive cardiac invalidism in spite of careful medical attention it was felt that total thyroidectomy should be tried.

*Operation:* February 20, 1934. Anesthesia: Local. Complications: The patient developed a positive Chvostek of a few days' duration only. No cord injury or frank tetany.

*Postoperative Course:* Two months after operation dyspnea, cough, precordial ache and palpitation had distinctly improved. The B. M. R. was minus twenty-six, the face and eyes had become markedly puffy, and thyroid one half and one quarter of a grain on alternate days was given. For fifteen months after operation the favorable progress continued; she was able to manage her house, walk one-quarter to one-half mile a day, be more active and feel better than in years. A B. M. R. in May, 1935 was minus sixteen. From June to September, 1935 the patient was moderately active with housework, but unable to walk outside because of increasing dyspnea and epigastric fulness. In October, 1935 she again entered a state of gross congestive failure with frothy sputum and edema of the legs. At present she is showing improvement on bed rest and diuretics.

*Result:* This patient has been distinctly improved and benefited by the operation. She and her family considered the result miraculous.

CASE 14. S. M. No. 335831. M. Aged thirty-four. Native. Unemployed. (Former shoe worker.)

*Diagnoses:* Rheumatic heart disease, mitral stenosis and regurgitation, aortic stenosis and regurgitation, auricular fibrillation, congestive failure.

*History:* This patient entered the medical service March 14, 1934. At the age of seven he had had chorea and at twelve, severe tonsillitis; there was no history of rheumatism. He was well up to five years before entry when he gradually developed dypsnea on exertion which had continued and become progressive. Three years before he developed a sense of fulness in the abdomen and marked palpitation. He entered a local hospital where he remained for two weeks being digitalized and receiving diuretics. The fulness in the abdomen subsided, but the palpitation continued. For two and a half years prior to entry he had been unable to work because of severe dyspnea and palpitation on walking even one block on the level. During this time he had cardiac asthma on four occasions. He slept on two pillows. He never had edema of the extremities. He was able to be up and about the house. He was taking digitalis one and a half and three grains on alternate days.

*Physical Examination:* The heart showed marked generalized enlargement, with apical systolic and diastolic murmurs, also basal systolic and diastolic murmurs with a systolic thrill; rhythm was grossly irregular with a rate of seventy. Blood pressure was 115/50. The lungs were clear. The liver edge was just palpable but nontender. There was no edema.

*B. M. R.* level was plus six.

*Rationale for Operation:* This patient had been quite incapacitated for any activity for two and a half years because of dyspnea and palpitation without gross edema. It was believed that operation would increase his general comfort and activity, and might permit a return to work.

*Operation:* April 4, 1934. Anesthesia: Local. Complications: None. Uneventful operative recovery.

*Postoperative Course:* Since operation, now eighteen months ago, this patient has done very well. Within six weeks he was able to walk a half mile without any palpitation and almost no dyspnea. He now walks two miles a day, has no palpitation, and has only slight dyspnea going up hill or over stairs. He takes care of the furnace and helps with housework. He is looking for a job, but has yet not found one. He sleeps well on one pillow. He has had no cardiac asthma. There are no signs of congestive failure. Two months after operation with a B. M. R. of minus twenty-one he was started on one quarter grain of thyroid every other day because of lethargy and sleepiness. He has continued on this dosage maintaining a rate about minus twenty-five without untoward myxedema symptoms. He is taking one pill of digitalis daily. His only present complaint is easy fatigability.

*Result:* This patient represents a very good result. His presenting symptoms have been almost entirely dissipated. He should be able to work when he finds a job which does not demand too strenuous activity.

CASE 15. S. D. No. 336686. F. Aged forty-two. Russian Hebrew. Housewife.

*Diagnoses:* Rheumatic heart disease, mitral stenosis and regurgitation, aortic stenosis and regurgitation, auricular fibrillation, congestive failure.

*History:* This patient entered the hospital for the first time April 23, 1934. Thirty years previously, at the age of twelve, she had rheumatic fever and was in bed for three months. Twelve years before she noted onset of palpitation and dyspnea which had continued and progressed since. Six years be-

fore she was forced to give up work in a restaurant because of severe dyspnea. Nine months prior to entry cardiac asthma first occurred and had been present almost nightly since. During this period the patient required three to four pillows for rest, and palpitation and dyspnea were so marked that activity was limited to a chair and bed life. She had been fully digitalized.

*Physical Examination:* The patient was sitting upright in bed with difficult breathing. The neck veins were engorged. The heart was enlarged to the left, showed double mitral and aortic murmurs, and was totally irregular with an apical rate of ninety. The liver was not enlarged. There was no edema.

*B. M. R.* level was plus eleven.

*Rationale for Operation:* It was believed that operation alone might offer further relief to this patient. Because symptoms of left-sided failure predominated without gross edema, it was felt that she should be a favorable subject, and while her activity might not be greatly increased, she should be made far more comfortable.

*Operation:* May 18, 1934. Anesthesia: Local. Complications: None.

*Postoperative Course:* This patient illustrates well some of the difficulties of postoperative management. For the first two months there was some symptomatic improvement, but no increase in activity; B. M. R. fell to minus nine. The rhythm had returned to normal. During July severe attacks of palpitation occurred associated with choking sensations, presumably paroxysmal fibrillation; dyspnea became as severe as before operation; B. M. R. rose to minus two and there were no signs or symptoms of myxedema. Catamenia was three weeks overdue and the question of possible pregnancy arose. An Aschheim-Zondek test was negative. In early August the B. M. R. dropped to minus twenty-eight, and mild signs of myxedema appeared, but the patient's general state was as poor as before operation.

August 21, 1934 the patient was readmitted to the house because of paroxysms of palpitation, nocturnal dyspnea, and several spells of syncope. She had taken one and a half grains of digitalis daily since the operation, as before. Electrocardiogram showed sinoauricular block with A-V. nodal escape beats, digitalis T. waves, and rate of sixty-five. It was felt that digitalis intoxication was in part responsible for her difficulties and this was omitted for a week and then resumed in one and a half grain doses daily. Because of marked appearances of myxedema (in spite of B. M. R. only minus twenty-two) thyroid, one quarter grain daily, was started. In September she was still confined to a chair and bed life. A normal catamenia occurred. Because of a pulse of fifty-five to sixty, digitalis was omitted every third day; thyroid was continued. In October conditions were much the same. In early November the patient appeared somewhat improved and was able to be up about the house about half the time; the pulse was down to fifty and the electrocardiogram showed digitalis T waves, so digitalis was reduced to one and a half grains every other day; the B. M. R. was only minus ten and the patient did not appear myxedematous so thyroid was omitted. In the middle of November the patient had two severe attacks of palpitation, dyspnea and choking sensations; the B. M. R. was minus twenty-two and the patient's face was quite puffy; pulse was fifty-six and regular; quinidine was tried to eliminate the paroxysms of fibrillation but after six grains marked intoxication occurred and the drug was omitted. In mid December the patient reported that there had been no further attacks of palpitation but that dyspnea had grown progressively more severe on the slightest exertion since thyroid had been omitted the month before, and that abdominal distention had become more troublesome; the B. M. R. reading was minus seventeen, but clinically it appeared to be in the minus thirties. With a pulse of sixty and no signs of congestive failure, we felt that the severe dyspnea, weakness and abdominal distention were most likely myxedema rather than cardiac symptoms. A seven foot chest plate at this time showed no change in the size or shape of the heart since operation. (We were wondering if she might show a change to the picture of a myxedema heart.) Thyroid one quarter grain daily was resumed. At the end of December the patient was much improved in every way, having much less dyspnea and distention and no other untoward symptoms; B. M. R. was minus seventeen.

From January until about the middle of May, 1935, she seemed improved, being able to help with the housework, having no palpitation, and very little dyspnea on the level. During this period her metabolism was about minus eighteen, on one quarter grain of thyroid daily, with a pulse of sixty to seventy on digitalis one and a half grains every other day.

From the end of May, 1935 up to the time of writing, October, 1935, sixteen months after operation, the patient has again failed, having some increase in dyspnea with walking on the level, palpitation when active or quiet, orthopnea requiring three to four pillows, and has been again forced back to a chair and bed life. In June she had a very profuse period with excessive flowing for nine days. She was readmitted to the hospital for observation. At this time the B. M. R. had risen to minus four (having been only minus twelve in May), and thyroid was omitted. Surgical consultants could find no evidence of pelvic tumor, and a course of x-ray over the uterus was given. There has been no menstrual flow since. In August the B. M. R. was minus nine, off thyroid, but since abdominal distention and general weakness had increased, it was felt wise to resume thyroid one quarter grain every other day. In September the B. M. R. was minus thirteen, weakness and distention were decreased, but dyspnea was not improved and several mild attacks of cardiac asthma had occurred.

*Result:* This patient's course has been one of ups and downs and complications. We do not feel that the operation has been worth while.

CASE 16. W. R. B. M. No. 14214. M. Aged sixty-seven. Canadian. Physician.

*Diagnoses:* Angina pectoris, coronary heart disease.

*History:* This patient entered the hospital June 4, 1934. Two years prior to entry he began to notice a pressure pain below the right clavicle appearing on exertion. For the six months before entry he had suffered very severe crushing precordial pains radiating down the left arm, coming on walking or any marked exertion, and always promptly relieved by nitroglycerine. The various purine drugs were of no value in preventing attacks. There were never any symptoms of congestive failure.

*Physical Examination:* The heart was at the upper limits of normal size. The sounds were of good quality and there were no murmurs. The lungs were clear. There was no edema. Blood pressure was 118/70.

*B. M. R.* level was minus seventeen. The electrocardiogram was normal.

*Rationale for Operation:* The patient was very anxious for the operation and the degree of disability from angina appeared sufficient for its recommendation in spite of the low basal metabolic rate.

*Operation:* June 19, 1934. Anesthesia: Local. Complications: None.

*Postoperative Course:* July 20, 1934, one month after operation, the B. M. R. was minus thirty-one. August 24, 1934 the rate was minus thirty-seven and there was some puffiness of the eyes; thyroid one quarter grain daily was started. In November he started doing some light work and reported that he had had no true angina since operation, but slight soreness over the precordium on exertion. When seen in September 1935, fifteen months after operation, he appeared to be very well, reporting only a little substernal aching on smoking or on walking any distance, but this was promptly relieved by nitroglycerine. His B. M. R. was minus seventeen, but recently he had been taking one grain of thyroid daily, as on this he felt better, although substernal oppression and aching had increased with this dosage. He was advised to reduce this to one half grain daily. He had been able to carry on with his office practice steadily except for a three weeks' vacation.

*Result:* This patient has shown almost complete relief of his anginal symptoms and represents a very satisfactory result.

CASE 17. M. S. No. 335249. F. Aged 45. Colored. Housewife.

*Diagnoses:* Rheumatic and hypertensive heart disease, aortic stenosis, mitral regurgitation, congestive failure.

*History:* This patient entered the medical service May 16, 1934 for the second time in congestive failure. There was no past history of rheumatic infection. Fifteen months before she had had a large hemoptysis and then learned for the first time that she had heart trouble. Following that time there was progressive dyspnea and palpitation on exertion, and a constant cough. For six months there had been localized precordial aches and pain over the region of the liver, and during the same period she had slept poorly with three pillows. Three months before February, 1934, she had remained for three weeks on the medical ward with mild congestive failure and was digitalized. Following discharge, in spite of continued bed rest and digitalis, she shortly developed more dyspnea, constant liver pain, nausea, edema of legs and cardiac asthma occurring every other night. She had done no work for fifteen months and had been a chair and bed invalid for six months.

*Physical Examination:* There was a moderate degree of jaundice. The heart showed marked enlargement with apical and basal systolic murmurs and thrills. The pulse was ninety and regular. The blood pressure was 180/124. The lungs showed moist râles at both bases. The liver was enlarged, edge palpable ten cm. below the costal margin and definitely tender. There was a small amount of abdominal fluid present. The legs and feet showed pitting edema.

*B. M. R.* level was plus twelve.

*Rationale for Operation:* Because of progressive cardiac failure, producing marked discomfort and total incapacity, in spite of rest and digitalis, it was felt that total thyroidectomy alone might offer relief.

*Operation:* June 10, 1934. Anesthesia: Local. Complications: None.

*Postoperative Course:* Following operation for about eight months this patient did well. She was up and about the house all day, got one of the meals, and walked as much as half a mile. She had moderate dyspnea only on going over stairs. She slept well on two pillows. Cardiac asthma ceased except for two slight attacks associated with respiratory infection. The chronic cough ceased. There was no longer any palpitation, precordial ache, liver pain or edema. Examination showed few râles persisting at the lung bases. Enlargement of liver persisted but the tenderness disappeared. Seven months after operation the B. M. R. fell to minus thirty-five, previous levels having been about twenty-six. Because of some increasing fatigue, very poor appetite and marked puffiness of the face, thyroid one-quarter grain every other day was started. One month later B. M. R. was minus twenty-four and the patient was feeling generally much improved. Nine months following operation cardiac asthma, dyspnea, palpitation and edema returned in increasing degree until ten months after operation the patient was chiefly confined to bed. Digitalis and thyroid was continued as before, B. M. R.'s ranging from minus twenty to minus thirty. On August 19, 1935, fourteen months after operation, the patient died in gross congestive failure at another hospital. No autopsy was obtained.

*Result:* For nine months this patient showed some moderate but distinct improvement. She then again grew progressively worse and died in gross failure at the end of fourteen months. We do not consider this result worth while.

*Comment:* The rapid recurrence of the second attack of gross failure, within three months of the first, with the patient remaining practically at bed rest on digitalis, suggests a severity of heart disease and a rapidity of failure which should have been considered a contraindication to operation.

CASE 18. W. M. No. 337153. M. Aged thirty-six. Unemployed.

*Diagnoses:* Rheumatic heart disease, mitral stenosis and regurgitation, auricular fibrillation, congestive failure.

*History:* This patient entered the hospital for the second time in congestive failure July 10, 1934. There was no past history of rheumatic infection. Two years before, following a hernia operation, he first noticed increasing dyspnea, palpitation and cough. One year before he had a hemoptysis. Following that there was orthopnea requiring three pillows at night, increasing attacks of cardiac asthma coming three or four nights a week, and numerous smaller hemoptyses. Two months before, he entered the hospital for ten days' treatment. Full digitalization produced slight improvement, but shortly after discharge symptoms all became more severe with marked liver pain, nausea, vomiting and the onset of jaundice. He had been unable to work for two years and had led a bed and chair life for six months.

*Physical Examination:* There was a marked degree of jaundice, orthopnea and cyanosis. The heart was grossly enlarged with apical systolic and diastolic murmurs; rhythm grossly irregular, rate eighty. The liver was felt five cm. below the costal margin and markedly tender. There were râles at both lung bases. There was no edema of the legs. Blood pressure was 120/70.

*B. M. R.* level was minus four.

*Rationale for Operation:* On full digitalization and bed rest we had seen this patient grow progressively worse over a period of two months. He was totally incapacitated and very uncomfortable from his severe cardiac asthma. It was felt that operation alone might offer him some relief.

*Operation:* August 10, 1934. Anesthesia: Local. Complications: None.

*Postoperative Course:* Since operation, now fourteen months ago, the patient has remained somewhat more comfortable than before, but has been able to increase his activity very little. He has spent about half of the time in bed, and only on rare occasions has been able to walk out to the street. Dyspnea and palpitation have continued to be severe on any but the slightest exertion. Cardiac asthma, which had caused great discomfort several nights a week for about a year prior to operation has completely ceased, except for several attacks associated with severe respiratory infection. While the B. M. R. fell to minus twenty-seven one month after operation, no marked signs of myxedema appeared until the fifth month when the rate was minus thirty-five. Uncomfortable myxedema symptoms have been controlled on one quarter grain of thyroid every other day. In the fifth month symptoms of digitalis intoxication appeared on one and a half grains daily, and since then the patient has been carried on five, one and a half grain pills a week.

*Result:* The patient feels that the operation has been worth while for the simple reason that cardiac asthma has ceased, however he has shown no improvement otherwise and is still a cripple. Operation has doubtless prolonged his life, but to what avail? In a situation of this type we cannot feel from our point of view that the procedure has been justifiable.

*Comment:* The comment of the previous case (No. 17) likewise applies here. Because of too rapid progression and too severe heart disease, this patient should not have been considered favorable for operation.

CASE 19. B. R. No. 337309. F. Aged thirty-three. Syrian. Stitcher.

*Diagnoses:* Rheumatic heart disease, mitral stenosis and regurgitation, aortic stenosis and regurgitation, congestive failure, hypertension.

*History:* This patient entered the hospital for the second time December 17, 1934. Twenty years before she had had acute rheumatic fever. For one and a half years she had noticed increasing fatigue on exertion. Nine months previously she first had several spells of paroxysmal nocturnal dyspnea. These were followed by some increasing dyspnea and palpitation, and two to three pillows were required at night. She was kept in bed for one month and digitalized. Following this, weakness and palpitation made it impossible for her to work. In May, 1934, seven months before the second admission she entered the hospital for study. While there total thyroidectomy was decided on, but the patient developed mumps and was discharged to return at a later date. Following another two weeks in bed at home she gradually started increasing her activity, but on doing housework she had a return of palpitation and dyspnea, and had several more attacks of nocturnal dyspnea. Symptoms continued about the same up to the time of entry and she was able to be up and about but not able to carry on any work.

*Physical Examination:* The patient was lying on two pillows in no distress. The neck veins were slightly full and pulsating. The heart was grossly enlarged to the left; with sounds of poor quality. There were aortic and mitral diastolic and systolic murmurs. Blood pressure was 184/80. The lungs were clear. The liver was not felt and there was no edema.

*B. M. R.* level was plus twenty-five.

*Rationale for Operation:* Because of slowly progressive and incapacitating cardiac symptoms in this patient under careful medical management, it seemed wise to give her the benefit of operation. The lack of any gross congestion or rapid progression seemed favorable factors, as did the consistently high B. M. R. (It was not felt that she had any thyrotoxicosis as was later proved by histologically normal gland.)

*Operation:* January 18, 1935. Anesthesia: Local.

*Postoperative Course:* Three days after operation the patient developed a positive Chvostek and Trousseau requiring calcium by mouth. The operative scar showed moderate drainage and there was some swelling in the neck. The cardiac compensation was satisfactorily maintained. Two and a half weeks following operation the patient suddenly developed what appeared to be pneumonia with some collapse at the left lung base. There were marked cyanosis and respiratory distress requiring an oxygen tent for two days. Following this there continued to be signs of congestion at the lung bases, the patient had to sleep on three to four pillows. There were marked general weakness and some edema of the sacrum and legs. On March 13, about two months following operation, further signs of consolidation appeared in the left lung, the temperature and pulse rose and the patient died in collapse.

*Autopsy:* Limited to the chest. There was no evidence of thyroid or parathyroid tissue found in the neck. The left lung showed a small area of atelectasis in the lower lobe, and there was scattered pneumonic consolidation through both lungs; there was no evidence of embolus. The heart was markedly enlarged weighing 725 grams. The aortic valve showed thickening and nodularity. The mitral valve showed no marked abnormality although there was slight thickening of the free margins. There was evidence of chronic myocarditis with small areas of fibrosis and a few small areas of round cell infiltration. The coronary vessels were normal.

*Result:* Because of postoperative pulmonary complications this patient was never out of bed following her operation. Doubtless aggravated by the infection, her cardiac decompensation increased.

*Comment:* This patient should have done well, but for uncertain reasons, complications arose and she never left the hospital.

CASE 20. H. R. No. 340719. M. Aged twenty-eight. Native. Unemployed.

*Diagnoses:* Rheumatic heart disease, mitral stenosis and regurgitation, aortic stenosis and regurgitation, congestive failure.

*History:* This patient first entered the hospital October 16, 1934. At the age of eleven, seventeen years before, he had acute rheumatic fever and for the following seven years was troubled by joint pains off and on. Following an appendectomy three years before entry, he developed a severe cough with the raising of some bloody sputum, and had never since been normally strong. A year and a half before entry he first experienced difficulty in breathing while at work, and shortly after started having paroxysms of nocturnal dyspnea. Nine months before entry he suffered a sudden attack of dyspnea, crushing, localized precordial pain, and hemoptysis. He was taken to a local hospital where he was digitalized. Following this there were localized precordial pain, coming in paroxysms not related to exertion, and not relieved by nitroglycerine; marked dyspnea with hurrying on the level or go-

ing over stairs; almost constant palpitation; and a constant productive cough. He required about three pillows at night. At the time of entry he had been unable to do any work for over six months, could not go over stairs and could only walk about one quarter mile on the level.

After a period of three and a half weeks' study in the hospital, during which time total thyroidectomy was decided upon, the patient developed an acute respiratory infection and was sent home to return for operation at a later date.

January 22, 1935 the patient was readmitted. For about six weeks he had been somewhat more symptom free than before his first entry, but ten days prior to entry he had another hemoptysis and attack of severe dyspnea requiring a hypodermic and local hospitalization. After four or five days of bed rest he was again improved.

*Physical Examination:* There was no cyanosis. The neck veins showed moderate pulsation. The heart was grossly enlarged to the left with sounds of poor quality; mitral and aortic systolic and diastolic murmurs were present. The blood pressure was 154/46. The lungs were clear, the liver edge was not felt, and there was no edema.

*B. M. R.* level was minus six.

*Rationale for Operation:* Because of symptoms largely of left-sided failure without edema, it was felt that this patient should do well after thyroidectomy. He had received adequate medical care and in spite of this remained incapacitated.

*Operation:* January 28, 1935. Anesthesia: Local. The day following the operation the patient developed a small collapse at the right base which cleared promptly. There were no other complications.

*Postoperative Course:* February 9, 1935 the patient left the hospital. Within one month his B. M. R. dropped to minus eighteen and he gradually began to increase his activities without discomfort. He was enabled to go over stairs and within two months could walk three or four miles a day without dyspnea. He had no paroxysmal dyspnea, almost no palpitation, and only occasional mild precordial ache. At the end of three months he had a B. M. R. of minus twenty-nine and appeared quite myxedematous; one quarter grain of thyroid every other day was started. At this time he seemed practically symptom free, slept well on one pillow and was going out trout fishing. In the fifth month he noted a distinct increase in lethargy, he was not able to concentrate or think so well as before, yet the B. M. R. had risen to minus eighteen. He noticed a distinct increase in dyspnea on any exertion, and was requiring three to four pillows at night. Another episode of severe precordial pain, severe dyspnea and hemoptysis requiring hypodermic and local hospitalization occurred. From that time on all of his cardiac symptoms were as severe as before operation and he had the added symptoms of marked mental dullness, drowsiness, irritability and abdominal distention in spite of a B. M. R. of minus twenty. Suddenly, about seven and a half months after his operation, the patient was seized by another severe attack of precordial pain, dyspnea and hemoptysis, and died within one hour of admission to his local hospital in spite of venesection and morphine. No autopsy was obtained.

*Result:* For a period of three to four months following operation, this patient appeared to be remarkably improved. Then, however, symptoms returned and the patient was quite miserable, finally dying with acute pulmonary edema seven and a half months after operation. While the operation may have afforded some temporary benefit, we do not feel that it was worth while.

*Comment:* The possibility of pulmonary thrombosis as a complicating factor in the case is to be considered as a possible explanation for the rather rapid downhill course.

CASE 21. L. H. B. M. No. 8126. F. Aged twenty-seven.

*Diagnoses:* Rheumatic heart disease, mitral stenosis and regurgitation, paroxysmal auricular fibrillation, congestive failure.

*History:* The patient entered the Baker Memorial Hospital May 2, 1935 for the fifth time because of heart failure. At the ages of twelve, fourteen, and seventeen she had had attacks of acute rheumatic fever. November 1932, March 1933, and October 1933 she had been admitted because of severe attacks of paroxysmal auricular fibrillation. June 1933 the patient was in the hospital for acute appendicitis with operation successfully done under spinal anesthesia. Signs and symptoms of definite congestive failure first appeared nine months before the present entry when increasing dyspnea and edema of the legs was noted at night, and palpitation became quite constant. Eight months before entry a chronic cough set in. Four months before entry she remained in bed for six weeks and under full digitalization edema disappeared. While in bed at this time sudden pain developed in the right side of the chest and the patient coughed up several clots of blood. On resuming moderate activity edema gradually returned and increased until three weeks before entry she was again forced back to bed. For nine months she had required three to four pillows at night.

*Physical Examination:* The patient was sitting up straight in bed. The heart was grossly enlarged to the left with apical systolic and diastolic murmurs, rhythm grossly irregular. Blood pressure was 150/80. The lungs showed slight dullness at bases. (Small amount of fluid by x-ray.) The liver was felt three to four cm. below the costal margin. There was moderate sacral edema.

*B. M. R.* level was plus fourteen.

*Rationale for Operation:* It was felt that this patient who was able to maintain compensation at bed rest, but for whom even slight activity was too much, should be a favorable case for operation, since she was young, had no complicating factors and a plus B. M. R.

*Operation:* May 13, 1935. Anesthesia: Local. Complications: None.

*Postoperative Course:* At the time of discharge, three weeks after operation, B. M. R. was minus three, heart consciousness and orthopnea were largely relieved. Two weeks after leaving the hospital the patient again began to develop edema requiring three more weeks in bed and salyrgan. For another two weeks she started to move about slowly, but again became edematous. Following that she was confined to bed with massive anasarca which could not be controlled by salyrgan up to the time of her death October 30, 1935, five and a half months after operation. About two months after operation marked coldness, swelling of eyes and lacrimation set in, making the patient uncomfortable. First one quarter grain of thyroid was given every other day, then daily and finally after about three months the dosage was raised to one half grain daily because of continued general discomfort from myxedema. B. M. R.'s were not done, but at the lowest, metabolism was judged to be about minus thirty. During the months following operation hemoptysis associated with severe chest pain occurred on several occasions.

*Result:* The patient showed no benefit from operation and continued a rapid downhill course.

*Comment:* This patient almost certainly had pulmonary infarction which was likely in large degree responsible for her rapid downhill course. Again she showed a rapidity of progression in the nine months prior to operation similar to that of cases 17 and 18 which should have made the procedure seem unadvisable.

## DISCUSSION

In the treatment of hopeless, progressive cardiac disease the physician grasps at any therapy which may offer relief to his patient. Such therapy, however, must offer more than the prolongation of life for a few weeks or months of distressful existence. Our experience with total ablation of the thyroid gland in the treatment of congestive heart failure seems to show that a certain prolongation of life was all that we had accomplished in some cases. In others, however, striking symptomatic benefit was obtained.

The trial of the procedure was designed to give us a point of view concerning this therapy as it could be developed in a general hospital, for it is doubtful if any method of treatment can become of far-reaching application that cannot be administered in any well integrated hospital.

It is in the problem of the selection of cases and the unpredictability of results in the individual case that the great obstacle to the recommendation of the operation lies. The list of contraindications to the operation has been given and attention to this in our clinic has finally resulted in a classification of cardiac patients in which all but a very small group are considered unsuitable for the procedure. It is extremely difficult to select a patient who on the one hand is not too sick to be benefited by operation, or on the other hand so relatively well that the physician may entertain fears lest the operation which he recommends may leave his patient worse than before. He must realize that, generally speaking, failure to relieve the patient by operation results in leaving on the physician's hands not only a cardiac problem, but also one of avoiding the discomforts peculiar to myxedema. Theoretically it should be easy, by means of a small dose of thyroid, to avoid the symptoms of myxedema. In actual practice, however, it has been our experience that even when the metabolic rate is maintained at levels where gross manifestations of myxedema are absent, there may yet remain certain unpleasant symptoms fairly attributable to the low metabolic rate. Even here nice adjustment of thyroid dosage may overcome the difficulties.

It is significant that while we have not abandoned this procedure, with the large number of cardiac patients seen on the wards in the past six months, in no case have we felt sufficiently confident to recommend the operation.

Our results in congestive failure indicate that in cases well selected and managed, worth while results may be expected in about half. With this in view, in a suitable case, it is for the physician and for the patient to decide whether a procedure of this magnitude, carrying with it certain discomforts and certain risks, is worth while for a temporary improvement in the course of an inevitably fatal disease. This is a point where individual philosophy must enter the picture.

Concerning the results in angina pectoris our series does not warrant the drawing of conclusions. In one of the two cases the effect of total thyroidectomy has been definitely beneficial. In our clinic intractable angina pectoris is more frequently treated by paravertebral alcohol injection than by thyroidectomy.

Recent figures from the Beth Israel Hospital, Boston, where the greatest amount of this work has been done, based on patients operated upon from one and a half to three years ago show the following results which are somewhat more favorable than ours[7].

TABLE 7

| | Congestive Failure | Angina Pectoris |
|---|---|---|
| Great Improvement | 12 (35%) | 18 (50%) |
| Moderate Improvement | 10 (29%) | 6 (17%) |
| Slight Improvement | 7 (21%) | 7 (19%) |
| No Significant Improvement | 5 (15%) | 5 (14%) |
| Total | 34 Cases | 36 Cases |

In addition to the cases listed above there were six operative deaths. We may reasonably assume that the cases showing "Great and Moderate Improvement" were worth while. This gives 64 per cent in the congestive failure cases and 67 per cent in the angina cases. Were the six operative deaths included in this table under "No Improvement" as in our table 4, the net results would be slightly lower.

From the Peter Bent Brigham Hospital, Boston, the other hospital in which this work was started, we are given the following results based

TABLE 8

| | | |
|---|---|---|
| CONGESTIVE FAILURE | 25 CASES | |
| Great Improvement | 6 | Considered Worth |
| Moderate Improvement | 5 | While Results |
| Slight Improvement | 2 | 9 Cases or 36% |
| No Improvement | 12 | (2 operative deaths) |
| ANGINA PECTORIS | 29 CASES | |
| Great Improvement | 11 | Considered Worth |
| Moderate Improvement | 10 | While Results |
| Slight Improvement | 4 | 19 Cases or 65.5% |
| No Improvement | 4 | (2 operative deaths) |

on cases operated upon from three years up to one year ago[8]. During the past year only three total thyroidectomies for heart disease have been done.

Three of the above cases were classified under both angina and congestive failure. These figures include the early cases operated on which were desperately sick and later realized to have had too severe heart disease to have been properly chosen for operation. In this series a case was considered a good result if physiological cardiac improvement occurred, as the case of a patient with angina having daily pain before operation and none for two weeks after operation but dying at the end of that time with a coronary occlusion; yet, as in this case, the patient might be classified as not showing a worth while result when the case is considered as a whole.

If further investigations permit more precise recognition of the criteria for the selection of cases and can give the profession a greater assurance of favorable results than it has at present, then certainly this form of treatment may be considered worth while.

## CONCLUSIONS

1. We are reporting the results of total ablation of the thyroid gland in twenty-one cardiac patients operated on at the Massachusetts General Hospital between July, 1933 and May, 1935. Nineteen patients had congestive failure and two had angina pectoris. Our conclusions are, therefore, based almost wholly on our experience with congestive failure.

2. Fifteen patients are now dead. In about one-fourth of the entire series the operation was considered worth while, in three-fourths it was not.

3. The relatively poor results in our series depend to a considerable degree upon the difficulty in selection of cases and in the fact that too severe cases were originally chosen. The contraindications to operation are numerous, but in cases well selected and handled, worth while results were secured, at least temporarily, in fifty per cent.

4. There is a small group of patients with cardiac failure in whom medical therapy is ineffective in controlling the progressive loss of cardiac reserve for whom total thyroidectomy offers an even chance of worth while improvement.

5. Avoidance of the grosser manifestations of myxedema we have not found difficult. Small daily rations of thyroid usually accomplish this purpose. In some cases, however, at a metabolic level above that of complete myxedema, low rate symptoms have been troublesome.

6. It is our belief that the procedure must be further studied before its usefulness in the treatment of heart disease can be fairly evaluated.

We are indebted to Dr. H. L. Blumgart for frequent advice and suggestions in the course of this study.

## REFERENCES

1. Blumgart, H. L.; Levine, S. A., and Berlin, D. D.: Congestive heart failure and angina pectoris; therapeutic effect of thyroidectomy on patients without clinical or pathologic evidence of thyroid toxicity. Arch. Int. Med. **51:** 866 (June) 1933.
2. Means, J. H.: Address on certain aspects of pathogenesis of angina pectoris. Canad. M. A. J. **24:** 193 (Feb.) 1931.
3. Lerman, J.; Clark, R. J., and Means, J. H.: Heart in myxedema; electrocardiograms and roentgen-ray measurements before and after therapy. Ann. Int. Med. **6:** 1251 (April) 1933.
4. Lerman, J.; Clark, R. J., and Means, J. H.: Further observations on heart in myxedema. Ann. Int. Med. **8:** 82 (July) 1934.
5. Levine, H. B., and White, P. D.: Personal communication in preparation for publication.
6. Berlin, D. D.: Therapeutic effect of complete thyroidectomy on congestive heart failure and angina pectoris in patients with no clinical or pathological evidence of thyroid toxicity; operative technique. Am. J. Surg. **21:** 173 (Aug.) 1933.
7. Blumgart, H. L.: Personal communication. (Nov.) 1935.
8. Levine, S. A.: Personal communication. (Nov.) 1935.

## MEDICAL AFFAIRS IN CONNECTION WITH THE CALIFORNIA PACIFIC INTERNATIONAL EXPOSITION

Tuesday, May 26, has been designated as California Medical Association Day at the 1936 California Pacific International Exposition, San Diego, which opened February 12 and will continue to September 9.

Approximately 3,000 physicians and their families are expected to visit the world's fair in Balboa Park upon that occasion.

The focal point of the conclave will be the Palace of Medical Science, one of the principal exhibit palaces of the 1936 exposition, where extensive displays will be sponsored by the leading medical societies and manufacturers of pharmaceutical supplies.

A program appropriate to the day is being arranged by an exposition committee of the San Diego County Medical Society, with Dr. Lyell C. Kinney, 1831 Fourth Avenue, San Diego, acting as chairman. Direct coöperation is being lent by Dr. Ben F. Eager, Medico-Dental Building, San Diego, president, and Dr. W. C. Crabtree, Medico-Dental Building, San Diego, secretary of the San Diego county society.

A medical exhibit in the Palace of Medical Science will be sponsored by the San Diego County Medical Society with the coöperation of the California Medical Association and the American Medical Association. Dr. W. H. Geistweit, Jr., of San Diego, has been named director of medical exhibits to arrange details of this exhibit.

It is predicted that the medical exhibits at the 1936 exposition will be the most extensive yet displayed. The whole Palace of Medical Science will be devoted solely to exhibits treating on the prevention and treatment of disease, and it will form one of the important features of this world's fair which is dedicated to mankind's progress in all realms of science, art and industry.

# NEW ENGLAND SURGICAL SOCIETY

## THE CONTRIBUTION OF THE COMMUNITY HOSPITAL TO BETTER MEDICAL SERVICE*

BY PEER P. JOHNSON, M.D.†

WHEN we compare the limited field of medicine of the "horse and buggy" days with the great progress of the present, the question arises as to whether the application of our art has kept up with its development. In the opinion of many it has not. Perhaps one of the most serious criticisms is that economic factors prevent a large proportion of our population from receiving suitable medical care. Other criticisms relate to free competition, the sliding scale, fee-splitting, the "appalling" incompetence of the family doctor, too much surgery, and the inability of the public to determine the qualifications of the doctor.

As a means of overcoming the economic difficulties, group practice or state medicine financed by taxation or insurance, either voluntary or compulsory, has been suggested. As a solution, group medicine may offer interesting possibilities of decreasing slightly the cost of medical care, but one can conceive of its being applicable to only a small number of communities. Even so, the quality of its service would naturally depend on the type of individuals making up the group. The incompetent would still be incompetent, the fee-splitter would still be a fee-splitter, and group competition would merely be substituted for that of the individual.

State medicine would undoubtedly provide medical care at a lower cost, but it is hard to understand by what magic it would improve the quality. Nothing in the experience of the countries which have adopted this form of medical practice suggests that a higher type of service is given. With the great amount of free service provided by doctors, hospitals and communities, it is difficult to believe that in New England there is much suffering because of the lack of adequate medical care.

It is unnecessary to elaborate to any degree upon the economic aspect of this subject for it is one which has been discussed in the lay and medical press, as well as in every recent medical meeting. The problem does exist and it behooves us to familiarize ourselves with its details and to give it our sympathetic consideration and coöperation to the end that the best interests of the patient and the doctor may be served.

Whatever solution is evolved, the medical aspects of the problem should be under the control of physicians and absolutely free from the domination of politics or of any organization operating for profit.

Most of the criticism emanates from economists, sociologists and committees investigating the cost of medical care, and this latter group seems to be made up of men who have but little professional contact with the so-called low income group. There is, on the other hand, no great evidence that the public itself is particularly dissatisfied with the quality of the service which it receives. An enlightened public can demand through its legislature, as it does not in Massachusetts, that only properly qualified physicians shall be permitted to take examinations for licensure; that those desiring to specialize shall give evidence of fitness and suitable training; and, further, if it so desires, that some form of examinations be held at reasonable intervals to test their competency and their right to continue in practice. These are matters over which the medical profession itself has no control. Even attempts on the part of our medical societies to discipline their members who are guilty of unethical practices raised the cry of oppression at the hands of organized medicine. Any attempts on our part to elevate the standards meet with immediate opposition. To relieve our profession of a source of serious and just criticism, some method is needed by which the fee-splitter, the man who operates for the fee alone, and the hopelessly incompetent can be eliminated.

While we may not yet have a satisfactory solution for the economic situation, there is still much that we, and the hospitals with which we are connected, can do to improve the quality of both medical and surgical practice.

At present the general practitioner finds himself between the devil and the deep blue sea. By some he is told that he should be able to diagnose and care for from eighty to ninety per cent of all illness; while by others that his incapacity is so great that he must give way to a new order. It is true that our changing economic life has placed him at a great disadvantage, and it is equally true that many of the newer diagnostic measures may be beyond his power to perform. The value and importance of these measures can certainly be understood by him and the indications for their application recognized. As with most of us, the mistakes which he makes are not so much due to his failure to make use of this bewildering array of diagnostic aids as to his failure to take careful

*President's Address delivered at the Annual Meeting of the New England Surgical Society at Manchester, N. H., September 27, 1935.

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histories and make careful examinations and carefully coördinate his findings. The tendency of the day is to rush for the diagnosis to the x-ray and the laboratory, aids which in a goodly percentage of cases should be merely confirmatory and supplementary.

I believe that the general practitioner is not so incompetent as his critics claim. I feel sure that he today gives a higher grade of careful, devoted, intelligent and self-sacrificing service than ever before. Whatever his shortcomings, it is obvious that he will still be in demand for long years to come. To what other agency could the acutely ill turn for relief? He should be the first to be consulted. His care may not necessarily, as of old, include the whole family, and it is quite probable that many of his patients will, and often to their disadvantage, consult specialists of their own selection.

In the smaller cities and communities, the hospital should provide him with the facilities for obtaining the various laboratory investigations and specialized assistance in the care of his patients, and this at a reasonable cost. Further, the hospital should provide him, if competent and ethical, with a place where he may treat his private patients. On the other hand, the hospital should limit the doctor to the type of work for which he is fitted by training and experience and should compel him to observe its high standards, so that a patient who enters its doors, whether charity or pay, shall know that he will be in competent hands. It is true that opening the doors of the hospital to larger numbers of doctors will complicate the administrative problem, but the benefits to both the doctor and the patient will far outweigh its disadvantages.

The great need of the physician is the continuance of his medical education in order that he may keep fully abreast of the times, and his failure to accomplish this is undoubtedly the principal cause of criticisms directed against him. As a rule, postgraduate medical education is available only by trips away from home and the expenditure of time which it is difficult to give.* Yet the hospital of his community contains an abundance of clinical material which could be used for teaching purposes. Hospitals have contented themselves with the care of the sick and the training of nurses but, from an educational point of view, have neglected their opportunities to be of service to the profession as a whole. The physician who feels that he cannot go away for postgraduate instruction will benefit as much by spending an hour or two each week in the hospital of his neighborhood. In this way he will not only get an opportunity to see a greater variety of cases than he would daily meet, but will come in closer contact with his fellow practitioners.

Firmly convinced of the value of this postgraduate teaching, we have, in our hospital, made available to the family doctor all our facilities, including autopsies. Many years ago we instituted a weekly operative clinic, but later changed to a monthly medical and surgical clinic followed by luncheon. That these clinics are considered of value is attested by the fact that there is usually an attendance of some thirty or forty physicians; that luncheon is not the principal inducement is evidenced by the fact that but a small number remain for it. More recently, in addition to the monthly clinic and the regular staff meeting, there has been instituted a weekly ward visit, followed two days later by an operative clinic. Rather than the usual perfunctory ward visit we have tried to make this a ward clinic, at which a small number of instructive cases are considered. Each one of these is turned over to a physician, who is told as briefly as possible the reasons for which the patient sought relief and is supplied with whatever information he desires as to the history, examination and laboratory findings. He is requested to discuss the case from the point of view of diagnosis and treatment, after which the whole group is encouraged to take part in the discussion. At the operative clinic, patients seen in the ward and requiring operation are presented, so that physicians interested may not only have a hand in the diagnosis but also see it verified or disproved, and at their subsequent visits observe the outcome.

Whether or not this employment of clinical instruction is the cause, it is rare indeed for a serious surgical emergency to be received from the hands of the family physician too late for operative relief. The signs of cancer are more early recognized and cases of perforated appendix have become less frequent. So suspicious has the doctor become of acute abdominal pain that only one in three of the patients entering the hospital with a diagnosis of acute appendicitis is such, and requires operation. This is, as least, a much safer course of action than the former one of procrastination.

Such clinics require leadership, time and effort, but the benefits derived are well worth the effort, for unconsciously the standards of the profession and of the hospital will be raised to the profit of the community.

The general practitioner and general surgeon are the offspring of the old family doctor. Their interests are identical. The diagnostic skill of the general practitioner determines in a large measure our surgical results. It is, therefore, our duty as a surgical organization to

*In Massachusetts, under the auspices of the Massachusetts Medical Society, postgraduate study has been furthered by courses of lectures given throughout the various districts by qualified specialists. The attendance at these lectures indicates that many physicians are anxious to take advantage of the opportunities for further study. The value of this method would be greatly increased if it could be combined with the use of clinical material.

work for the betterment not only of the surgeon but of the general practitioner, of whom Sigerist, in his preface to "The Great Doctors," has fittingly said: "Bach and Mozart would be dead for ever, were it not for the living artists who are perpetually reviving their melodies. Pasteur and Koch would have lived in vain but for the everyday practitioners through whose activities their teachings are made effective. It is not so much the great theoreticians upon whom the health of the community depends, as the huge army of family doctors who succor the ailing from hour to hour."

Surgery should certainly be practised only by those thoroughly trained in the art. Our first President, Doctor Mixter, in his presidential address, speaking of the surgeon, said: "It must be strongly emphasized that the surgeon is not born but made. Thorough training of the mind and hand, hospital experience under the control of able masters—these are the absolute essentials and should be insisted on in the future as they have not been in the past."

What Doctor Mixter said nineteen years ago is even more true today. With the further advances in surgery, the general surgeon's field of activity has been broadened and he therefore requires an even longer and more thorough period of training. Under our present system this necessary training has been acquired chiefly by the surgeons who practice in the large cities, but, unfortunately, the same high standards do not prevail in the smaller communities. Surgery here is too often done by the occasional operator whose preliminary training, in many cases, has consisted only of one year's interneship in some small hospital and possibly some few months of postgraduate study. The surgical material of these communities is divided among so many men who feel qualified to do surgery, that no one has an opportunity to acquire the judgment, diagnostic skill, dexterity, speed and confidence that the best interests of his patient demand. In other words, there are too many men doing surgery and too few real surgeons. Our present system develops mediocrity in many rather than ability in a few. It is fair to say that where now a dozen or more men do the surgical work of a small hospital, two or three thoroughly trained surgeons with a competent house staff could, to the advantage of the community, do it better. For it is a well-recognized truth that as a surgeon's experience increases, not only his mortality but his morbidity rate decreases. But how will he acquire this experience if he is not kept constantly employed?

There are in New England some 300 hospitals. In twelve small hospitals, taken at random, there were 14,290 operations done last year. In some of these, treating from 1500 to 3000 patients, the operations were done by fifteen or more operators. In one alone with less than 1500, fourteen men did 300 major operations. And such a proportion is probably the rule rather than the exception in a large number of these institutions. In two of these hospitals studied twenty per cent of the operations were appendectomies, and both hospitals had large surgical staffs. They removed three times as many appendices as another hospital treating the same number of patients but with a surgical staff limited to three. Using these figures on the appendix alone, would it not be logical to consider that decreasing the number of surgeons would decrease the number of unnecessary operations?

Although this organ is a potential source of danger, it is not the cause of every acute belly-ache and its needless removal is hardly warranted. In spite of its advances, surgery has not reached that stage of perfection where the abdomen can be opened without risk. Except under the most unusual circumstances, I do not feel able to agree with the statement of Dean Lewis that the general practitioner should do his own appendectomies. And as a reason one need only reflect on the difficulties in diagnosing an obscure case of this disease as well as the complex problems with which he must often deal when operating for what at the outset appears to be a typical case of appendicitis.

Another factor of equal importance in preventing the development of the general surgeon is the length of his hospital service. Such services are often for one, two to rarely more than three months. One small hospital in the above list has four members on its surgical staff who serve in rotation through the year of periods of one month. They hardly become acquainted with their patients or get their stride before they pass off the scene. No one can possibly conceive that under such a scheme the patient will get the highest type of care. In hospitals with services of two or three months, the surgeon is left unemployed for the remainder of the year except for such private work as may come his way. If he happens to have a large surgical practice such an arrangement is of no detriment, but it constitutes a serious letdown for the young surgeon. As a result of this his surgical development is definitely retarded to the disadvantage of his community. In practically no other important line of endeavor would one expect to reach a high degree of excellence with such limited opportunities.

In this matter of length of service there seems to be a unanimity of opinion, but little effort is made to alter the situation. The problem has been discussed with many surgeons, and with but one exception all have held the opinion that a much longer period of hospital service, or even better, a continuous service would be the ideal arrangement. All agreed that rapid turnovers

were bad for the patient and that their own judgment and skill were greater at the end than at the beginning of their services. Their opinions might be summed up in that of one surgeon who wrote: "I feel that the longer the service the better. I should consider it ideal to have the service continue the year round, but with enough men on duty to divide the work without its being an excessive burden on any individual. Personally, I am on a two-months' service. At the end of the two months I feel relatively quite competent. During the ten months off duty I feel that I am getting rusty, with a rust that cannot be readily polished off even by visiting other clinics. As far as physical ability to stand the gaff is concerned, I feel again that a longer period of less strenuous activity is less fatiguing than a short, overburdened one. From what observation I have been able to make, the best surgery is done in hospitals with longer services." From the very nature of things, the accomplishment of this ideal implies the division of surgery among a smaller number of surgeons.

As the surgical standards of the community hospitals are raised, there will be less necessity for patients to go to the large medical centers, often not only a hardship to them and their families, but an increased expense. The peripatetic surgeon, too, except in the rare and more difficult cases, will find himself less needed. His position at best cannot be entirely satisfactory either to himself or to the patient. Unable, owing to the distance from his base, to conduct a period of observation, he often feels compelled to operate when it would be wiser to wait. Under the most favorable circumstances he is obliged to leave the postoperative care to the local physician who may or may not be able to carry the patient through a stormy convalescence.

Because all surgery is done there, the hospital is the only organization which can limit the physician to the field for which by training he is qualified. Its surgical staff should be selected only on the basis of training and ability and in a number sufficient to the surgical needs of the community. Certainly the staff should be small enough to give each man an opportunity to maintain a high degree of excellence.

If the objection to this is that it limits free competition in the surgical field, the answer is that the interest of the patient is paramount. If a limited number of surgeons can reduce the mortality, morbidity and unnecessary operations, with the resulting economic consequences, no further argument is needed. There is no justification for allowing everyone, who so desires, to do surgery. The amount of surgery is of necessity limited, and if, by confining the field to a few well-trained men, the results are better, the personal desires of the many must be sacrificed. Probably the one factor which, more than anything else, keeps the highly trained young surgeon in the big city is the knowledge that he will be unable to find a field in the smaller community and will have to content himself with general practice as a means of livelihood, while his surgery becomes a side issue.

It may well be that at some future date, for the best interests of the patient and the hospital, surgery will be limited to a small staff on an adequate salary. These men would give their full time to the hospital and would be relieved of the unpleasant duty of collecting fees from the patient. Under such circumstances the reasons for fee-splitting, unnecessary operations and operations for the fee alone would be removed.

Hospitals have been willing to spend large sums of money for buildings and equipment but too little for human material. Here is an opportunity to make use of that great army of well-trained young doctors who are unemployed and crowding the big cities. They could be utilized as the resident house staff without which no hospital, however small, can really operate at its highest efficiency. This association with young men, fresh from the schools and hospitals, would be of mutual advantage. It would stimulate the older men to keep up-to-date, while it would provide the younger men with many of the practical aspects of our art with which they are still too unfamiliar.

For our small communities the hospital should be the center of medical activity. With well-equipped laboratories, providing service at a reasonable cost, with a well-trained staff under intelligent leadership, with its doors open to the competent and ethical general practitioner for the treatment of his patients, it would provide a satisfactory substitute for group medicine in which the doctor would not lose his individuality, and would answer the public's question of how to obtain a competent physician.

# EMOTION AND DIARRHEA*

BY ALBERT J. SULLIVAN, M.D.†

THE purging effect of strong emotion has been recognized for centuries. In this paper, the early medical views on simple diarrhea are reviewed, observations from the literature on this psychomotor phenomenon are presented, and recent work on the etiological relationship of emotion to certain diarrheas, usually considered organic in origin, is discussed. A plea is made for more thorough investigation of this "no man's land", the field of psychosomatic relationships.

## NERVOUS DIARRHEA

Alvarez, in his excellent book on "Nervous Indigestion", considers the ways in which emotion can affect the gastrointestinal tract. He reminds us that references to the purging effect of fear or anxiety may be found as early as 700 B.C. when Sennacherib, in describing his battle with two young kings of Elam, noted that, "Like young captured birds they lost courage. With their urine they defiled their chariots and let fall their excrements." In Genesis, 43:30, we read that, "Joseph made haste for his bowels did yearn upon his brother, and he sought where to weep." In Caxton's edition of Aesop's Fables is found, "The wulf shate thryce for the grete fere that he had." So well known is the direct association of strong emotion and defecation that many references to it can be found in folklore and in various profane authors, both classic and modern.

The scientific interest of the medical profession in this phenomenon seems to have appeared at a much later date. Sydenham[1] in his Epistolary Dissertation in 1682 wrote, "Hysteria on the stomach will create continued vomiting; on the bowels, diarrhea." The effect of the psyche on the colon seems to have concerned Van Swieten[2], for among other cases he cites this one: "I have seen a man who had taken a sufficiently nauseating draught, not only shudder and be nauseated, but also be frequently purged, when he merely saw the cup in which he had taken the medicine." In the medical literature of the nineteenth century there are many references to the effect of mental states on the bowels, particularly the purging effects of placebos and bread pills when they were considered by the patients to be cathartics. Tuke[3] cites two interesting examples of the effect of the imagination on the intestinal canal and adds his own amusing comment.

> "In the 'Bibliotheque choisie de Medecine' is a good example of the effect produced by the Imagination, during sleep, upon the action of the intestines. The daughter of the Hanoverian Consul, aged 18, having to take a rhubarb purge on the following day, which she especially disliked, dreamed that she had taken the hated dose. Griped by her imaginary rhubarb she awoke, and the bowels acted freely five or six times. Precisely similar is a case which I give on the same authority (Demangeon); that of a monk for whom some purgative had been prepared, to be taken on the following day. He dreamed that he swallowed the medicine; the consequence of which was that he was aroused by the necessity of attending to the calls of nature, and was copiously purged eight times. All must admit that any medical man who would engage to insure the same operations from imaginary as from real rhubarb or senna, would enjoy a fashionable purgative practice."

About the middle of the nineteenth century the relationship between emotion and diarrhea received general recognition by the medical profession and nervous diarrhea became an accepted diagnosis. Habershon[4], in 1857, listed ten causes of diarrhea, the ninth in the list being "Marked agitation or fright." John Chapman[5], in 1866, devoted a whole chapter to "Diarrhoea Originated by the Mind." The following paragraph is quoted verbatim from that chapter:

> "The following facts I am able to authenticate: (1) A woman who has a drunken husband suffers great anxiety when he is away from home, especially if late at night, lest anything should happen to him, and particularly lest by a fall or other accident he should receive bodily harm. This anxiety brings on diarrhoea, accompanied with trembling, pallor, and a peculiar haggardness of countenance.
>
> "(2) A lady who, while crossing the Atlantic, suffered fearfully from sea-sickness and violent diarrhoea during the whole passage, has since her marriage been troubled with diarrhoea almost always when she has experienced painful emotions. On almost every occasion when her husband is unkind to her, as he is wont to be, she has a violent attack of diarrhoea.

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Read before The National Society for the Advancement of Gastroenterology, New York, October 23, 1935.

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"(3) A woman who suffers from diarrhoea whenever her feelings are vehemently excited, even although the excitement may be one of sudden pleasure.

"(4) One of my patients, who was reading George Eliot's noble work 'Romola,' assured me that the emotions it excited in her brought on diarrhoeas! In fact, owing to this remarkable transformation of emotions into 'motions', she was obliged to abstain from reading the book for a time.

"(5) A lady, one of my patients, when affected by any violent emotion, especially if of a distressing character, is almost immediately attacked with diarrhoea, or vomiting, or both.

"(6) A gentleman, one of my patients, already mentioned, always finds himself attacked with diarrhoea after he has experienced any considerable mental excitement. It is well known, moreover, that many soldiers especially young ones, are attacked with diarrhoea when going into action."

Such a remarkable series of cases indicate that Chapman was years ahead of his time in the investigation of psychosomatic relationships. Unfortunately, most of his work was soon forgotten because of his peculiar therapeutic notions. He insisted that not only diarrhea and cholera but also epilepsy, paralysis, uterine affections and seasickness would be cured by the application of his special spinal icebags which was the "only available power of subduing hyperaemia of the automatic nervous centres". Today he might be considered a quack but it is rather interesting that he secured a high percentage of cures in a series of diseases which are frequently psychic or functional by a method which was probably effective because of its strong psychotherapeutic appeal.

Although all this material and much more, was available in the literature, many of the standard medical works of the nineteenth century and even later dates make no mention of nervous diarrhea. Neither Bennett's "Clinical Lectures" nor Ziemssen's 16 volume "Cyclopaedia of the Practice of Medicine" mention the subject. On the other hand, Trousseau[6], the great French clinician, devotes much space to the discussion of this form of diarrhea. He says, "It was necessary, Gentlemen, that I should enter into these details, because nervous diarrhoea is one of the most frequent forms of the affection, and is at the same time one of those in which the physician can be most useful, when he knows how to recognize it."

Toward the end of the nineteenth century, the concept of nervous diarrhea began to be widely recognized. In the first (1892) edition of Osler's "Practice of Medicine"[7] we find the following discussion:

"It is by no means clear how mental states act upon the bowels, and yet it is an old and trustworthy observation which everyday experience confirms that the mental state may profoundly affect the intestinal canal. These influences should not properly be considered under catarrhal processes, as they result simply from increased peristalsis or increased secretion, and are usually described under the heading nervous diarrhoea. In children it frequently follows fright. It is common, too, in adults as a result of emotional disturbances. Constatt mentions a surgeon who always before an important operation had watery diarrhoea. In hysterical women it is seen as an occasional occurrence, due to transient excitement, or as a chronic, protracted diarrhoea which may last for months or even years."

Inasmuch as this discussion concerns only the clinical aspects of the problem, I shall not attempt to review the work of modern investigators such as Cannon and Alvarez to whom we owe so much for their important physiological investigations in this field. An excellent concise review of the literature on psychic influencing of gastrointestinal secretion and motility is given by Alvarez[8].

## ORGANIC DIARRHEAS

So far we have considered diarrhea only as a symptom of emotional episodes. None of the group of diarrheas commonly called "organic" have been considered. In the foregoing quotations the close association between cause and effect (the emotion and the diarrhea) has not escaped the physician and the diagnosis of nervous diarrhea was fairly obvious. The diarrhea was short and lasted for the duration of the emotion (usually fear or anxiety). When presented with a case of chronic diarrhea lasting one or more years, the true relationship between cause and effect was blurred and it is easy to see how possible emotional origins were rarely traced. A chronic diarrhea is rarely painless, as long-continued irritation results in other symptoms referable to the colon. It thus takes on the aspect of an "organic" disease. When the physician can find blood, pus or mucous casts in the stools and the patient exhibits constitutional signs of organic disease such as fever and leukocytosis, it is almost impossible for the organically-minded physician to realize that an emotional episode may have initiated the "disease" no matter how distant its beginning, or that chronic emotional disturbances may have been responsible for the chronic diarrhea.

Before we consider the early historical references concerning the effect of emotion on so-called "organic" diarrheas it is well to discuss briefly the rough classification of organic diar-

rheas as they were described in the middle of the last century. Most diarrheas characterized by bloody stools (whether they occurred in epidemics) were called dysenteries. Epidemics of cholera were seen, even in England. However, sporadic cases of diarrhea, especially if sudden in onset, with copious liquid stools and accompanied by fever and vomiting were nearly always diagnosed as cholera. The chronic diarrhea associated with pulmonary tuberculosis was usually recognized as tuberculous enteritis. Mucous colitis had been described under a variety of names. Ulcerative colitis was first described by Wilks in 1859[9]. This description was amplified by Wilks and Moxon in 1875[10] and by White in 1888[11]. However, in the earlier literature undoubted cases of this disease can be found under the name of "simple intermittent dysentery"[12]. It is interesting, in view of our present idea that the disease is psychic in origin, to find that one of the earlier synonyms for the disease was "asylum dysentery".

It is, of course, confusing to find the terms diarrhea, dysentery and cholera used almost synonymously, therefore certain reservations must thus be made in the interpretation of some of the quotations which will be mentioned.

The earliest reference I have been able to find indicating that emotion may initiate an "organic" diarrhea is in Habershon[4]. In chapter 10, "On Colitis and Dysentery" he reminds the reader that "the depressing effect of night watching, and of witnessing the rapidly fatal termination of the disease, tends also to induce the complaint". Chapman[5] in his discussion of the causes of cholera writes as follows:

> "That fear, fright, and panic are fruitful sources of cholera is attested by a large number of authoritative witnesses. There is an old, and often repeated story of an encounter outside an Eastern city between the plague demon, when about to enter the city, and a citizen who asked what he was going to do there, and who was told by the demon that he was going to kill 3000 people. On his return from the city, the same citizen taxed him with lying, inasmuch as 30,000 had been killed. True, said the demon, but I only killed 3000; fear killed the rest." This story contains a great truth, which is recognized by almost every observer of a cholera epidemic. Referring to the last epidemic in Turkey, Mr. Harry Leach says, "Panic undoubtedly increased its intensity in many instances, and brought cases of simple diarrhoea into the grip of cholera very speedily indeed." Drs. Bell and Stokes remark, "many have been destroyed by fear alone"; and Dr. Wood says, "Sudden and strong emotions, often bring on an attack." Dr. Macpherson observes, "Cholera attacks all who are to be seized, and confidence seems to be of use both in warding off an attack and in struggling through it. The excessive alarms during epidemics are most injurious." Dr. MacCormack says he can assert from personal experience that "fear alone will produce the disease", and makes the following statement (often made also by others) for which, however, he gives no authority. "In the year 1832, a man was unfortunately tempted, by a large sum, to occupy for a certain time a bed in which he was informed a cholera patient had died; and although such had not been the case, he nevertheless from pure fear, was in a very short time seized with the symptoms of cholera and died." Dr. Forbes Winslow pithily remarks, "During an attack of cholera, the patient who has the least fear of dying has, coeteris paribus, the best chance of living."

Tuke[3] gives us several more examples of the emotional origin of "cholera".

> "The story of the Russian convicts under sentence of death, some of whom were placed in beds falsely said to have been occupied by cholera patients, will occur to the reader. Mr. G. Smith reported in the *Lancet* of August 4, 1866, the case of a fine hale blacksmith under surgical treatment in King's College Hospital, who carried down the bed on which a cholera patient had died. He sat up until late brooding over what he had done and its probable consequences. He died next morning of cholera. Those, however, who believe that cholera is contagious would not admit that, in this case, Fear was more than the exciting cause of the attack.
>
> "When, some years ago, the cholera was prevalent at Newlyn, a fishing village near Penzance, intercourse was forbidden between the two places. One day a man entered the shop of a barber in Penzance, and was shaved. On leaving, some one, who had recognized him, asked the barber if he knew whom he had been shaving. He replied, he did not. "Why, he's a man from Newlyn!" It was enough. The terrified barber was seized with cholera, and died within twenty-four hours.
>
> "Mr. . . . . of Falmouth, some years ago, had the cholera. When well, he went to the Lizard for a change. The woman who opened the door of the house to which he went, having heard that he had had the cholera, was exceeding alarmed, and had an attack herself."

### MUCOUS COLITIS

The foregoing material has, I believe, indicated two things: (1) that hyperactivity of

the gastrointestinal tract as expressed by diarrhea is frequently the result of certain strong emotions and that it has been recognized by laymen and physicians for many years, and (2) that certain physicians have felt for nearly a century that occasionally strong emotion was the precipitating factor in certain cases of diarrhea ordinarily considered "organic".

How have we, as physicians, made use of these two concepts? The first, that of nervous diarrhea, has received general recognition. Most of us, who have had classmates seized with severe diarrhea at examination time or have had comrades in the trenches so affected before going "over the top", have remembered in our practice that there was such a syndrome as nervous diarrhea. All too often, however, it has been a diagnosis by exclusion after we had vainly searched for all other possible causes, or perhaps the correct diagnosis has been presented to us by the patient who insisted that it was "just nerves". Not very often do we search for emotional disturbances in our patient's life as systematically and conscientiously as we do for amebae in his stools.

The second concept, that emotion may be responsible for initiating an "organic" diarrhea, has been almost completely ignored. Let us consider that syndrome which is usually spoken of as mucous colitis. The term is a misnomer, of course, for "mucus" in the stools is not invariably present nor necessarily a prominent feature nor is "colitis" applicable since there is ordinarily no inflammation of the colon. It is not a true diarrheal disease since constipation is, at times, a prominent feature. However, it serves well to illustrate a few points that deserve emphasis.

I will not go into the historical aspects of the disease (which are of interest chiefly because of the thirty-five different names which have been applied to it) other than to state that it had been thoroughly investigated and described by American clinicians long before it received the critical consideration of foreign physicians. It was first described by Mason Good[13] as "Diarrhoea Tubularis—Tubular Looseness."

In discussing mucous colitis, Osler[7] writes, "The cases are almost invariably seen in nervous or hysterical women or in men with neurasthenia. Mental emotions and worry of any sort seem particularly apt to bring on an attack. In mucous colitis no benefit can be expected from remedies administered by the mouth . . . the general nervous condition should receive appropriate treatment."

Since that time many writers have expressed similar opinions. However, the "organicists" die hard. In one of the leading textbooks[14] of medicine of today, we read, "Mucous colitis was formerly regarded as a neurosis, but clinical and pathological observations now justify the conclusion that it is a form of chronic colitis." Yet the description of the disease continues in these terms: "It frequently, though not invariably, occurs in patients with enteroptosis. Spastic constipation is always an accompaniment, it is far more common among women than men, and the sufferers are often highly neurotic!"

Here is a syndrome suspected for years by medical men, of being emotional in origin yet rarely treated as such. Practically the only thorough case reports are in the psychiatric literature. The articles by medical men are largely attempts to prove a bacterial, an allergic or a deficiency origin of the disease. It is amusing (or rather, tragic) to note how often such a study is prefaced by the remark "in an attempt to place this disease on a scientific basis". Many commendable attempts at the study of this syndrome have stalled at the halfway mark and "neurogenic colopathy" has been added to our terminology. Our old friend "vagotonia" and "sympatheticotonia" are used to explain the syndrome. Just because emotions make use of the vagal and sympathetic nerve pathways, does a syndrome become "neurogenic" instead of "psychogenic"?

Our insistence on finding an organ (the "soma" as opposed to the "psyche") on which to pin the cause of a syndrome has led us astray before. If we are to be consistent, now that the autonomic nerve centers have been discovered in the diencephalon, we should discard vagotonia and sympatheticotonia and speak of "diencephalonopathy". But if it is discovered that the pituitary gland is the activating agent of these centers we are then back on the trail again, tracking down adrenals, thyroid and gonads. We must not forget the merry chase we have had to find the organ responsible for diabetes.[15] First the kidney was responsible for spilling sugar, then Bernard's sugar puncture gave us a localistic neurogenic concept, then Mehring and Minkowski with extirpation experiments shifted us to the pancreas. Banting and Best reinforced this concept, but soon it was obvious that liver, muscles and other organs were involved. Recent investigations show that a depancreatized animal can live if the pituitary is also removed. Has the trail only led us back to the neurogenic concept of Claude Bernard? Remember that in this disease, also, there have been many who have thought that occasionally the disease was psychogenic.

And to return to our unfortunate patients with mucous colitis, how do they fare? Teeth, tonsils, appendix, and gallbladder are removed as foci of infection. Prostate and tubes are under suspicion. Bacterial flora are subjected to cultivation by a form of intestinal gardening. A belt or even an operation for a mobile cecum or ptosed transverse colon may be advised. Colonic irrigations are not so popular

since we have learned that they may result in a true colitis where only a questionable one existed before. But how frequently do we sit down and talk over with the patient his fears and hopes, his joys and sorrows? The answer is discouraging.

### ULCERATIVE COLITIS

We now come to a disease, nonspecific ulcerative colitis, which until recently has always been considered bacterial in origin. Superficial comparisons of autopsy material from this disease with that from bacillary or amebic dysentery would immediately suggest a bacterial or protozoal etiology. I shall not go in detail into the evidence supporting either of the two leading schools of thought, one believing that the disease is an attenuated dysentery and the other that it is due to a specific diplococcus. Each group can find bacteriological and experimental evidence to support its stand. For therapeutic results you can take your choice, for each group reports good results in seventy-five per cent of the cases, one with polyvalent antidysenteric serum[16] and the other with antidiplococcus serum[17]. Others have obtained the same results with transfusions[18] or mercurochrome, intravenously[19].

Have gastroenterologists always been satisfied that the disease was bacterial in origin? By no means, for other etiological agents have been suggested such as vitamin deficiency or disturbance in calcium metabolism. In 1923, Logan[20] came to the conclusion that bacteria were secondary invaders of the walls of the colon. He suggested that changes in the digestive juices of the upper gastrointestinal tract or in the endocrine secretions were responsible for the local lowering of tissue resistance. He said, "It thus seems probable that a general metabolic disturbance is responsible for the trouble." In 1925 Thomas R. Brown[21] wrote, "Is it not possible that the cause of the disease is to be found not in the presence of a definite and specific infective agent but rather in the absence of some protective substance or mechanism of something which normally inhibits the bacterial invasion of the intestinal wall, perhaps due to metabolic error, or endocrine disturbances or lack of specific bacteriophage, or absence of some normal bactericidal substance in the intestinal mucosa."

To the late Dr. Cecil D. Murray[22, 23] belongs the credit for demonstrating that this disease may be psychogenic in origin. Interestingly enough, this was discovered only because ulcerative colitis was included in a group of disorders in which it was thought desirable to investigate psychogenic factors. Murray writes[23], "The fact that the Constitution Clinic in the Department of Medicine was undertaking a special investigation of the psychological panel in certain disorders is the only reason why this and other patients were referred to us for psychotherapy. Although this patient was described as 'nervous', she did not complain of mental symptoms and would not have fallen into the group ordinarily referred to the psychiatric clinic." In 1930, Sullivan and Chandler[24] added five cases to Murray's twelve and gave a follow-up of one of Murray's cases. In 1935, Sullivan[25] added nine more cases and included follow-up studies of the entire group, some as long as four and a half years. Jones[26] and Daniels[27] have added other cases.

These studies show a striking unanimity in the underlying psychogenic factors. Primarily a disease of young adult life, it affects those with definite infantile elements in their emotional make-up who are unable to adjust themselves to the sexual and financial responsibilities of adult life. Nearly always there is a well-marked time relationship between emotional crises and the onset of the disease or its recurrences. The patients have many personality traits in common. Psychotherapy is astonishingly successful.

We now have partial answers to some of the problems which have frequently arisen in this disease. Why should it take its toll of youth in its best period, "Frequently the recent bride or the vigorous youth in his college years, or a young husband and provider"?[16] And we hope that an end has been put to such therapeutic nihilism as this[28]: "The writer has gradually reached the conviction that in the individual case the patient's course is somehow predetermined from the start regardless of therapy. This idea has come from the experience that some patients get well promptly under any form of therapy, whereas others seem totally refractory to all measures."

### SUGGESTIONS FOR FUTURE INVESTIGATIONS

In reading the literature on ulcerative colitis I have been struck by the brevity with which the social and psychological background of the patients has been treated. Whole pages of possible emotional conflicts have been indicated by such phrases as, "a widow of 23" or "an unmarried Jewish girl of 29". The study of the *patient* has ended with such a phrase, though the study of the *disease* would fill volumes. In only one case was enough of the patient included to enable us to say that psychogenic factors may have played a rôle in this instance. Trousseau[6] reports a case, "The patient stated that two years ago she was living in Champagne, when she was obliged by the pressure of poverty, to seek a home elsewhere. Accompanied by her husband and one child, the sole survivor of six, she came to Paris to seek a subsistence. In this attempt she failed and in place of obtaining the hoped for relief, she got involved in still deeper

misery. Her husband fell ill; she, he, and the child had nothing to eat save the ration of bread allowed by public charity." The diarrhea was of two years' duration, therapy was unavailing and the patient died. Autopsy showed nothing but superficial ulcerations of the colon.

The problem, as I see it, is this: Here is a disease which in some instances, at least, is emotional in origin. Are we going to continue to dismiss the patient and his problems with a phrase, the result of a few superficial questions? Are we going to neglect emotions in ulcerative colitis as we have in mucous colitis? Is it scientific to study only bacterial flora and immune reactions and mere nonsense to probe into the patient's psyche?

In order to demonstrate what I believe the true investigation of these psychosomatic disorders involves, I find it convenient to borrow illustrations from Crookshank[29] and Alexander[30]. Crookshank has written:

> "It always seems to me odd in the extreme that doctors, who when students, suffered from frequency of micturition before a viva voce examination, or who when in France, had actual experience of the bowel looseness that occurred before action, should persistently refuse to seek a psychical correlative—not to say an etiological factor—when confronted with a case of functional enuresis or mucous colitis. I often wonder that some hard-boiled and orthodox clinician does not describe emotional weeping as a 'new disease', calling it paroxysmal lachrymation, and suggesting treatment by belladonna, astringent local applications, avoidance of sexual excess, tea, tobacco and alcohol, and a salt-free diet with restriction of fluid intake; proceeding, in the event of failure, to early removal of the tear-glands. This sounds of course, ludicrous. But a good deal of contemporary medicine and surgery seems to me to be on much the same level."

Here, then, is the so-called "scientific" method of studying paroxysmal lacrimation, with chemical estimations of the salt content of the tears, and a correlation of this with the blood chloride level. Alexander[30] has supplied us with a description of the psychological method of studying such a disorder in the following quotation:

> "It can easily be shown, however, that even the most common psychomotor processes cannot be satisfactorily described without the precise knowledge of psychic factors. As an example, the process of weeping or laughing may be considered, both of which are based on complicated psychomotor reflexes. A statement that sad ideas are able to influence the function of the lacrimal glands is a vague generality, the scientific uselessness of which becomes clear from the following imaginary experiment. The problem is to establish those conditions under which the physiologic processes of weeping are provoked in different individuals. Let it be assumed that to solve this problem a hundred individuals are exposed to a moving picture. It will then be observed that by a certain touching scene a certain percentage of the hundred are unable to control their tears and react with the unsuccessful suppression of sobbing, whereas another group are much less touched and a third group remain entirely cold and observe critically the plot without any emotional participation in it. It would be, however, entirely false to jump from these differences of reactions to the conclusion that the persons who remained cold are less sentimental in general, because a second experiment will prove that, confronted with another scene on the screen, a great percentage of those who remained undisturbed by the first scene will now react with intensive sobbing and crying. If this kind of observation in exposing the experimental individuals to different scenes is patiently followed up, it may be possible to distinguish certain specific situations to which certain groups of individuals react with weeping and the specific sensitiveness of certain individuals to certain situations might even be considered as a characteristic feature of them. Experimentation may lead one thus far in this complicated field, but, if one wants to have a deeper insight into the intricate process of weeping, it will be necessary to investigate each individual separately. Such an individual psychologic study necessarily leads to very complex psychologic causal chains. In order to establish the specific sensitiveness of the experimental subjects to certain scenes, one must know their past life history, the whole development of their personality, because one will be able to understand the conditions under which an individual cries only from the experiences of his early life. The significance is now apparent of my former statement that the psychophysiologic process of weeping cannot be described by a general statement that certain sad ideas or impressions are able to influence the function of the lacrimal glands. If scientific demands regarding psychomotor processes are to be as strict as those accepted for somatic processes, the psychologic side of a psychomotor reaction has to be investigated with the same precision as is usual in studying organic processes."

If, in the foregoing illustrations, we substitute the colon for the tear-glands, and diarrhea for weeping we have an idea of how our problem should be attacked. Not for a moment do I believe that psychological studies will provide the answers to all our questions. There is much to be done on the somatic side; the mechanisms involved in the hyperperistalsis, the hypersecretion of mucus and the production of ulceration have still to be solved. There is plenty of scope for those investigators who will continue the work so ably begun by Cannon, Alvarez and Cushing. However, in the care of our patients there is an immediate need for physicians interested in the psychic investigation and treatment of such psychosomatic disorders. In this group are included not only diarrheal diseases but also essential hypertension, Grave's syndrome, gastric and duodenal ulcer and cardiospasm.[31]

## CONCLUSION

Material from the literature has been presented to illustrate the close relationship between emotion and diarrhea. An attempt has been made to show that diarrheal disease usually considered organic may be emotional in origin. A plea has been made for the substitution of thorough personality studies of these patients in place of our present "skillful neglect". Perhaps I may be allowed, in closing, to express two hopes.

First, that the present generation of physicians may keep in mind what was written by Hawthorne[32] in the "Scarlet Letter." "A bodily disease which we often think of as a thing apart and separate, may after all, be but a symptom of an illness in the spiritual part of our nature."

Secondly, that the coming generation of physicians will have been so taught in their medical curriculum that they will feel as Mohr[33] does, that, "There is no such thing as a purely psychic illness or a purely physical one, but only a living event taking place in a living organism which is itself alive only by virtue of the fact that in it psychic and somatic are united in a unity."

## REFERENCES

1. Sydenham, T.: The Works of Thomas Sydenham. London: Sydenham Society. 1848-50.
2. Van Swieten, G.: Commentaria in H. Boerhaave Aphorismos decognocendis et curandis morbis. 1775.
3. Tuke, D. H.: Influence of the Mind upon the Body in Health and Disease. London: J. Churchill. 1872.
4. Habershon, S. O.: Observations on Diseases of the Alimentary Canal. London: J. Churchill. 1857.
5. Chapman, J.: Diarrhoea and Cholera. London: Trübner & Company. Second Edition. 1866.
6. Trousseau, A.: Lectures on Clinical Medicine. Trans. of edit. 1868. London: The New Sydenham Society. 1871.
7. Osler, W.: The Principles and Practice of Medicine. New York: D. Appleton-Century Company. 1892.
8. Alvarez, W. C.: Ways in which emotion can affect the digestive tract. J. A. M. A. **92:** 1231 (April 13) 1929.
9. Wilks, S.: Lectures on Pathological Anatomy. London: Longmans. 1859.
10. Wilks, S. and Moxon, W.: Lectures on Pathological Anatomy. Second Edition. London: J. Churchill. 1875.
11. White, W. H.: On simple ulcerative colitis and other rare intestinal ulcers. Guy's Hosp. Rep. **30:** 131, 1887-88.
12. Harty, W.: Observations on the Simple Dysentery and Its Combinations. London: W. Smith & Son. 1805.
13. Good, M.: The Study of Medicine. Philadelphia. 1825.
14. Niles, W. L.: Colitis. In: A Text-book of Medicine. Edited by R. L. Cecil. Philadelphia and London: W. B. Saunders Company. 1930.
15. Dunbar, H. F.: Emotions and Bodily Changes. New York: Columbia University Press. 1935.
16. Crohn, B. B. and Rosenak, B. D: Follow-up of ulcerative colitis (non-specific). Am. J. Digest. Dis. & Nutrition **2:** 343 (Aug.) 1935.
17. Bargen, J. A.: Colon, Rectum and Anus. Rankin, Bargen and Buie. Philadelphia and London: W. B. Saunders Company. 1932.
18. Kantor, J. L.: Diarrhea. Am. J. Digest. Dis. & Nutrition **2:** 1 (March) 1935.
19. Andresen, A. F. L. and D'Albora, J. B.: Tr. Am. Gastroenterol. A. 36-48, 1933.
20. Logan, A. H.: Chronic ulcerative colitis cured by iodin. M. Clin. North America **7:** 105 (July) 1923.
21. Brown, T. R.: Chronic ulcerative colitis. Ann. Clin. Med. **4:** 425 (Nov.) 1925.
22. Murray, C. D: Psychogenic factors in etiology of ulcerative colitis and bloody diarrhea. Am. J. M. Sc. **180:** 239 (Aug.) 1930.
23. Murray, C. D.: A brief psychological analysis of patient with ulcerative colitis. J. Nerv. & Ment. Dis. **72:** 617 (Dec.) 1930.
24. Sullivan, A. J. and Chandler, C. A.: Ulcerative colitis of psychogenic origin: report of 6 cases. Yale J. Biol. & Med. **4:** 779 (July) 1932.
25. Sullivan, A. J.: Psychogenic factors in ulcerative colitis. Am. J. Digest. Dis. & Nutrition **2:** 651 (Jan.) 1936.
26. Jones, C. M.: Discussion[25].
27. Daniels, G. E.: Personal Communication.
28. Bloomfield, A. L.: Disease of the gastrointestinal tract. In: Internal Medicine. Edited by J. H. Musser. Philadelphia: Lea & Febiger. 1932, pp. 644-739.
29. Crookshank, F. G.: Organ-jargon. Brit. J. M. Psychol. **10:** 295, 1930. Quoted by Dunbar[15].
30. Alexander, F.: Functional disturbances of psychogenic nature. J. A. M. A. **100:** 469 (Feb. 18) 1933.
31. Moschcowitz, E.: The psychogenic origin of organic diseases. New Eng. J. Med. **212:** 603 (April 4) 1935.
32. Hawthorne, Nathaniel, 1850. Quoted by Dunbar[15].
33. Mohr, F.: Die Wechselwirkung körperlicher und seelischer Faktoren im Krankheitsgeschehen. Klin. Wchnschr. **6:** 772 (April 23) 1927. Quoted by Dunbar[15].

## THE PREVENTION OF DECAY OF TEETH

Science reports that decay of the teeth with attendant toothaches may be prevented if a method developed by Dr. E. P. Brady, of the Washington University Dental School, is put into successful practice. A dental examination can determine by a chemical test which of the teeth in one's mouth are liable to decay. Silver nitrate, a common drug used generally for germ killing purposes, betrays the presence on the tooth enamel of certain faults of formation. It is in these faulty areas that decay is likely to start, because there the acids in the mouth and acid-producing bacteria can penetrate through the enamel to the sensitive dentine beneath. The decay can be prevented by the use of silver nitrate. After it has started, its progress can be stopped by use of another chemical called trichloracetic acid, Dr. Brady said. This substance acts to coagulate the organic material in the tooth and block any further penetration by the acids of decay into the interior of the tooth. Nature has her own way of doing just this, under favorable conditions. When decay starts, a defense may be made in the form of a barrier of calcium deposit across the path of the penetrating acids.—*Science.*

# NEW HAMPSHIRE MEDICAL SOCIETY

## TREATMENT OF BURNS*

BY GROVER C. PENBERTHY, M.D.†

A REVIEW of the literature which refers to the treatment of burns indicates that the greatest amount of constructive thinking and work along lines of practical therapeutics has been done in the last two decades. It is also apparent that in spite of the advances made in the treatment of burns the general mortality remains high. No definite explanation can be given for this as there are many factors which contribute to the mortality. The care of the burn lesion still remains a serious surgical procedure. The ability of the human body to withstand the severe burn appears to be an individual problem, with the very young and the very old reacting poorly.

Statistics[1] show that about 45 per cent of the lethal burns occurring annually in the United States are in children under six years of age and in this group the outcome is often out of proportion to the severity and extent of the apparent damage done. At the Children's Hospital in Detroit, where 547 cases have been studied over the past twelve years, the greatest number fell in the age group one to three years.

The management of a burn case develops practical and theoretical considerations. Many questions have been raised and it is obvious that there are problems for further research before we can agree on any standard form of treatment, especially in the complicated cases.[2] There are advocates of the continuous saline bath[3]; the paraffin wax method[4]; the use of gentian violet[5], which has a decided advantage in minimizing infection, and many others.

In discussing the subject of burns it appears that up to the time that Davidson[6] brought out the use of tannic acid many of the clinics and physicians had adopted methods of their own and were satisfied with the results obtained. The oils, ointments, and other remedies were used with gauze dressings applied, which usually adhered to the burn area and caused pain and bleeding when removed. The extensive burn was usually given a bad prognosis, and the less severe case became infected, running a protracted course and developed deformities due to contracture.

The work of the late Doctor Edward C. Davidson is a contribution to medical science and it is with pride that we in Michigan refer to the results of his laboratory and clinical research. It was my good fortune to have the opportunity of working with him at the Children's Hospital[7], where the technic, which will be shown in the movie film, was perfected. His work has also had its effect in stimulating others to perfect methods of technic in the treatment of burns, which will assist in reducing the mortality. The advantages of the tannic acid form of treatment and the results obtained by its use have been emphasized in a number of published reports[8, 9, 10, 11, 12].

In referring to the use of tannic acid, Wells makes the following statement; "The introduction of tannic acid by Davidson in 1925 has revolutionized and apparently for the first time in history standardized the treatment of diffuse burns."

The early symptoms following a burn are primarily those of shock with a profound disturbance of the circulatory and heat regulating mechanism, and, in all probability, equally serious interference with many other normal functions of the body. The larger the area involved, the greater are the number of nerve endings and neurons irritated, resulting in a greater degree of shock. It has been shown by the experimental work of Davidson that a burn causes a marked depression in blood chlorides[13, 14] that the toxemia accompanying a burn is due to a toxic agent which originates at the site of the burn; that the absorption of this agent is responsible for the constitutional reaction; that the local destruction of tissue gives rise to a proteid substance with the subsequent formation of a proteose, and, finally, that the latter is the toxic element in burns.

Vogt[15] and later Vaccarezza[16] observed that when parabiosis was established between two animals and one was burned the other showed evidence of toxemia. It was further demonstrated that toxic symptoms did not develop in the unburned animal when it was separated from the burned animal within twelve hours, but that both animals finally died of toxemia when left united.

Pfeiffer[17] isolated toxic cleavage products of protein decomposition from burned skin. Robertson and Boyd[18] demonstrated the toxicity of the products of protein autolysis of burned tissue. They further showed that the toxin circulated in the blood stream and upon this was based their clinical work of exsanguination transfusion.

The clinical[6] and experimental facts suggest that the rational manner of combating the toxemia would be some form of local treatment

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which would prevent absorption from the site of the burn. This has been attempted in a number of ways. Débridement has deserved a certain amount of popularity, but its application is limited by the severity of the procedure.

Tannic acid is a nonnitrogenous amorphous powder readily soluble in water, glycerine and alcohol but insoluble in ether and chloroform. It precipitates proteins, alkaloids, some glucosides and the salts of heavy metals. It forms a more or less stable compound with the protein constituents of the body fluids and cells, thereby preventing the loss of body fluid at the site of the burn. The astringent effect appears to be limited to the superficial layers of tissue. The precipitated proteins on the surfaces treated, prevent and minimize the absorption of the autolytic products of protein decomposition. In addition, the precipitated proteins provide a protective coating and a mechanical action against sensory and inflammatory irritation. It is used with the idea of precipitating the toxic elements in the burned tissue, thereby preventing their absorption. Tannic acid is used as a five per cent aqueous solution freshly prepared and applied to the burned area by a DeVilbiss spray. A burn when exposed to the air is very painful and the earlier it is covered with tannic acid the degree of pain and shock will be greatly diminished.

The treatment of burns is best carried out in the hospital[19]. Patients admitted in shock can be put in a light tent or the heat may be applied in another way by an electrically heated blanket or many hot water bottles placed about the patient. Morphine or codeine should be given freely for the pain. This allows the patient to rest the first night and having had this rest he is better fitted to take the forcing of fluids the following day. One of the most essential features of the management of all burn cases is that of keeping up the fluid balance in the body[6]. For the more severe burns saline should be given by hypodermoclysis or intravenously with glucose as the needs indicate, in addition blood transfusion and this repeated as often as indicated.

The local treatment of burns depends to a large extent upon the experience of the surgical staff. The general rule, however, is to wash the acid and alkali burns with water before applying a neutralizing agent and to remove all devitalized superficial tissue, as well as opening and draining the blisters with as little trauma as possible. The patient is then placed in a light tent upon a sterile sheet and the tannic acid spray started by the nurse.

The burned area is sprayed every fifteen minutes for the first four to six hours at which time there is usually a brownish black firm leathery coagulum formed. The time required for this spraying depends upon the depth of the burn and the reaction of the tissues to the tannic acid. It is our practice to use the tannic acid jelly for burns of the face. The first application of the tannic acid gives the patient relief from pain and as the precipitate increases it becomes black and hard giving a firm protection which is a distinct advantage. As the epithelium regenerates, the coagulum or tannic leathery coat curls up at the edges.

It has been found in our use of the tannic acid in the treatment of burns in children that the mortality has been reduced from 36 per cent to less than 10 per cent. These figures compare very favorably with those reported from other clinics[10, 11].

The treatment of any burn case becomes an individual problem, but, to insure the best end-result, requires strict adherence to the accepted principles of treatment as proposed by Davidson and others. This treatment in the majority of cases should reduce the mortality and assist in preventing the unfortunate occurrence of many complications[2]. Prompt application of local treatment[20], proper supportive measures, follow-up care and attention together with early skin grafting, along with the correction of deformities, offers a reasonable hope for success.

## SUMMARY

(a.) Burns have carried a high mortality and are best treated in the hospital. The standardization in the treatment of diffuse burns, by the use of tannic acid, has materially reduced the mortality and this contribution to the subject by Doctor Davidson has resulted in the saving of many lives.

(b.) Administrations of a sedative upon admission.

(c.) Washing the chemical burns with water is essential before applying the neutralizing agent.

(d.) Proper first-aid followed by early hospitalization and aseptically cleansing the burned area is important.

(e.) The tannic acid method of treatment, combined with the light tent, is effective and helps to simplify the treatment of a condition that can tax the patience, time and resources of the attending physician to the utmost.

(f.) The use of tannic acid lessens the loss of body fluids and the coagulum acts as a protection against infection.

(g.) The forcing of fluids and giving saline solution, either subcutaneously or intravenously, and blood transfusion are necessary to carry the patient through the acute period of toxemia.

(h.) Early skin grafting of the third degree lesions will minimize the amount of scarring and deformity. It will also lessen the period of morbidity and disability.

(i.) The severe burn case often requires subsequent plastic surgery to improve the function of an extremity or better the cosmetic result.

REFERENCES

1. Pack, George T.: Thermic Injuries, Cyclopedia of Medicine. Philadelphia: F. A. Davis Company, 1934.
2. Penberthy, G. C. and Weller, C. N.: Complications associated with the treatment of burns. Am. J. Surg. **26:** 124, 1934.
3. Blair, V. P.; Brown, J. B., and Hamm, W. G.: Early care of burns and the repair of their defects. J. A. M. A. **98:** 1355. 1932.
4. Sherman, W. O.: Paraffin-wax treatment of burns. Surg. Gynec. Obst. **26:** 450, 1918.
5. Aldrich, R. H.: The rôle of infection in burns. New Eng. J. Med. **208:** 299, 1933.
6. Davidson, E. C.: Tannic acid in the treatment of burns. Surg. Gynec. Obst. **41:** 202, 1925.
7. Davidson, E. C. and Penberthy, G. C.: The treatment of burns in children with tannic acid. Detroit Proceedings Inter-State Postgraduate Medical Association of North America. P. 265, 1929.
8. Seeger, S. J.: The hydrogen-ion concentration value of tannic acid solutions used in the treatment of burns. Surg. Gynec. Obst. **55:** 455, 1932.
9. Wells, D. B.: The aseptic tannic acid treatment of diffuse superficial burns. J. A. M. A. **101:** 1136, 1933.
10. Wilson, W. C.: The tannic acid treatment of burns. Special Report Series, 141 Privy Council. Medical Research Council, London, Published by His Majesty's Stationery Office. 1929.
11. Stanley-Brown, Margaret: Extensive burns. Surg. Clinics of North America. **15:** 375, 1935.
12. Bancroft, F. W.: Cutaneous burns. Nelson's Looseleaf Living Surgery. Vol. **1:** P. 787.
13. Davidson, E. C.: Sodium chloride metabolism in cutaneous burns and its possible significance for rational therapy. Arch. Surg. **13:** 262, 1926.
14. Davidson, E. C.: The prevention of toxemia of burns: treatment by tannic acid solution. Am. J. Surg. **40:** 114, 1926.
15. Vogt, E.: Versuche über die Uebertragbarkeit des Verbrennungsgiftes. Ztschr. f. exper. Path. u. Therap. **11:** 191, 1912.
16. Vaccarezza, R. A.: Sur la cause de la mort par les brulures. Compt. rend. Soc. de biol. **86:** 1114, 1922.
17. Pfeiffer, H.: Experimentelle Beiträge zur Aetiologie des primären Verbrennungstodes. Virchow's Arch. f. path. Anat. **180:** 367, 1905.
Idem: Experimentelle Studien zur Lehre von den Autointoxikationen. Ztschr. f. Hyg. u. Infectionskrankh, Leipz. **54:** 419, 1906.
18. Robertson, B., and Boyd, G.: Toxemia of severe superficial burns. J. Lab. & Clin. Med. **9:** 1, 1923.
19. Penberthy, G. C.: Tannic acid treatment of burns. J. Michigan M. Soc. **34:** 1, 1935.
20. Penberthy, G. C., and Weller, C. N.: Treatment of burns at the Children's Hospital of Michigan. Brochure distributed at Scientific Exhibit, A. M. A., Cleveland, 1934.

## DISCUSSION

PRESIDENT LORD: This paper of Dr. Penberthy's on the Treatment of Burns with Tannic Acid will be discussed by Dr. Walter H. Lacey of Keene.

DR. WALTER H. LACEY: *Mr. President and Gentlemen of the New Hampshire Medical Society*—This paper constitutes a direct appeal simply put, to give our burn cases better care, especially the severe cases. The earlier you get the burns cleared up, the sooner you can graft, and the sooner you graft, the less the trouble will be, and this shows us the way.

It is interesting to note what some of the larger clinics in New England are using at the present time. The Children's Hospital in Boston is using tannic acid spray and contemplating using the tannic acid bath.

At the Massachusetts General Hospital, the tannic acid bath at 100° F. is being used and it is felt that this is an advance in treatment.

At the Johns Hopkins and New Haven Hospitals, gentian violet is being used and it is felt that it is more analgesic and keeps the lesions cleaner.

At the Presbyterian Hospital in New York, tannic acid compresses are being used.

All of these clinics, like ourselves, are bothered by the first aid treatment of greasy dressings applied outside which makes the initial clean-up at the hospital a little harder to do.

If we get the public and the nursing profession, as well as the doctors, to appreciate the value of these drying and tanning dressings, as outlined with such justifiable enthusiasm as Dr. Penberthy has, our burn cases will suffer less and get well more quickly. I thank you.

PRESIDENT LORD: I will call upon Dr. James W. Jameson of Concord for a further discussion of this paper.

DR. JAMES W. JAMESON: I think we owe a debt of gratitude to Dr. Penberthy for his excellent paper and for his very instructive illustrations of the tannic acid treatment for burns.

Since the work of Dr. Davidson on the treatment of burns with tannic acid, a great deal more thought has been given to this condition than ever before, with the result that the mortality in burns has been lowered and probably will be decreased further in the future.

The older forms of treatment with carron oil, various powders and ointments and things of that kind, without any regard to asepsis, employed just as a first aid and then continued, have made the mortality of burns very high, due especially to the factors which attend this serious condition.

The dressings which have been used in the past have been painful. They have required frequent changes; they have prolonged the period of disability and not only caused loss of time, but very definite economic loss.

Dr. Penberthy has covered these various factors which are the important ones in burns; first of all, shock, next the loss of body fluids, which someone has estimated that in the course of the first twenty-four hours, a burn covering one-sixth of the body will mean a loss of at least seventy per cent of the body fluid; thirdly, the absorption of the toxic products of the burn, and fourthly, the infection.

I feel that any form of treatment which will cut down the mortality, and which is simple in its use, is the one which should be used.

Gentian violet has been spoken of, and it has appealed to me very strongly for the simple reason that it has a bactericidal action, which, of course, tannic acid does not have.

The treatment of the serious burns, that is, the second and third degree burns, and those are the ones we are considering, is practically the same in any method which you use. That is, the shock must be combated, the loss of fluids taken care of by hypodermoclysis, and other methods of increasing the fluid intake, and the toxic products removed; but then as contrasting between tannic acid and gentian violet, you have one which produces a coagulum without any antiseptic property, and another which produces coagulum with an antiseptic property.

I have had no cases in which I have used gentian violet, but I have felt that it was a very satisfactory form of treatment theoretically. I should like to ask Dr. Penberthy about that and whether they have compared the treatment of gentian violet and the treatment of tannic acid.

The one difficulty which I have had in using tannic acid is in those cases where infection occurs somewhere in the middle of the tanned area, and fluctuation or tenderness cannot be found early;

then, after a period, in which the patient has been running an irregular temperature and has shown signs of sepsis, a definite abscess is finally found. This is probably due to improper preparation of the burned area before it is treated and occurs usually in the extensive deep burns.

I think that Dr. Penberthy has presented a fine paper to us, and it should be of great interest, not only to the medical profession, but to the lay public, because, as has been said the first aid treatment in these cases has oftentimes been the cause of a good deal of difficulty later on.

PRESIDENT LORD: This paper is now open for general discussion.

DR. THOMAS W. LUCE: I simply wanted to say that I own a book, which I think is a very valuable publication, because it was printed and published in Portsmouth in 1803. It was written by Dr. Pitman of London, who put out this edition in America. His purpose was to have it go into the homes of people for them to get suggestions and to know how to treat diseases. In this book, there is a chapter on burns.

Apparently, in that day, the preponderance of burns was in old ladies, who sat beside the fire. So he devotes quite a bit of that chapter in telling them that they should wear woolen skirts and not crinoline, and that they shouldn't open the door and scream but that they should lie down and roll over. Then he goes on to discuss the treatment of burns. He starts out by saying, "Never, under any circumstances, put grease on the burn" because it does no good but does harm, because it prevents the action of other remedies that are beneficial. Now, that, gentlemen, was in 1803. Dr. Jameson has spoken about the old methods but we find this pretty reasonable.

Then, continuing in the book, he says that in every household there should be a bottle of brine, in which there always were some sliced potatoes, so that the burn could be immediately covered with that solution.

The trouble I have with burns is that practically every burn I have to treat has been treated before I get it with lard and all that sort of thing. I think if that old book could be reprinted, at least that chapter on burns, and given to every family, it would be very helpful today.

PRESIDENT LORD: Is there further discussion on this paper of Dr. Penberthy's?

DR. HARRY E. MOCK: *Mr. President and Gentlemen*—I have heard Dr. Penberthy give this paper a number of times when he has talked on this subject of burns, and I am impressed with the kindness and the sincerity with which he always refers to the work of Dr. Davidson. We owe a great deal to Dr. Davidson for doing the early research work in tannic acid treatment of burns. But, I think the country over, and the profession, owes a great deal, if not the most, to Dr. Penberthy, for popularizing the treatment of burns with tannic acid. He is so modest about it himself that you would never know this from him, but I don't believe there is any man in the country who has done more toward bringing the mortality rate down, and relieving suffering from burns than has Dr. Penberthy.

I want to add to the list of hospitals that one of the speakers gave, St. Luke's Hospital in Chicago. Tannic acid is used there practically altogether in the treatment of burns.

I would like to emphasize one point, viz., the tent, left at an even temperature by means of an electric light bulb and allowing the patient to lie under this tent completely naked. This adds materially to the ease of caring for these cases.

And one point about the infection. Dr. Penberthy simply mentioned one case where they didn't do it over with the tannic acid because it was infected. Often when you get these cases, after two or three days at home, and then brought into the hospital, they are infected, and I would like to have Dr. Penberthy emphasize just what they do about putting tannic acid immediately upon such cases.

DR. ROGERS: Dr. Penberthy mentioned that eighty per cent of his burns were avoidable, also that about ninety-nine per cent were in the nonpaying group. You know the condition of the minds of people surrounding a child who has convulsions. Sometimes the child is put into too hot water by the parent.

I feel that as we have contacts with our patients, and as we have frequent opportunities to address public gatherings, we should speak on these dangers.

DR. ARMEN S. MANGURIAN: Tannic acid treatment is an old-fashioned treatment and we do not know it. When I was a young boy I recall burns being treated in this manner: One would gather leaves from the English walnut, or if these could not be obtained leaves from the oak tree. The leaves of the walnut are long, wide, and thin somewhat resembling those of the rubber plant. The leaves are boiled as one boils spinach, cooled, and spread over the burns. Both varieties contain a large percentage of tannic acid.

Tannic acid is also used in dyeing rugs if a good brown color is desired, the wool or yarn being boiled in the solution obtained from the leaves. The concoction derived from the leaves is also used as a hair dye, which gives a nice brown tint to the hair, acts as a tonic to the scalp and the roots. This vegetable dye is much better than the acid ones used in cosmetics in many countries.

Even now, I have found in my practice many people using old-fashioned remedies. I am much interested to find out their ingredients and have come to the conclusion that many of them contain the desired remedy and therefore, am never angry to find a patient using some old-fashioned remedy.

DR. PHILIP H. GREELEY: *Mr. President and Fellows*—I would like to add what I believe is a valuable prophylactic hint for the family or anyone of us who happens to come in contact with extremely hot water.

If cold water is dashed on to the injury at once the destructive cooking is checked immediately whereas if the assistant tries to remove clothing valuable time is lost and a bad injury results. As a personal experience helps to impress a valuable suggestion I will say that I have often heard my mother explain that she gave me this treatment when I sat down in a pail of near boiling water and saved me from serious results with a convenient pump and hasty action.

I believe if this could be broadcast into every home many a child might be saved serious injury.

PRESIDENT LORD: Time passes very rapidly; we can't seem to help it. I think that the evidences of interest in Dr. Penberthy's paper have been obvious this morning, and I will ask him to say a few words in closing.

DR. GROVER C. PENBERTHY: I wish to thank the discussants for their kind remarks. The subject of burns is of interest to all of us, and naturally the general practitioner must see more burns than we do in the clinics.

It is very interesting to hear the experiences of the other men. We all have our problems, and it is very apparent that we are all thinking along the same lines. It speaks for good medicine; it means

education of the public. I think Dr. Rogers mentioned the education of the public in the prevention of burns, which is very important. Since the laymen have an understanding of the importance of these matters, it is only a question of time when, perhaps, we can cut the mortality to a figure even lower than it is at present.

The matter that Dr. Jameson referred to regarding infection in the middle of the coagulum, I would say is apparent many times. Where the infection manifests itself, that coagulum, of course, should be cut, if not removed, in order to allow surgical drainage. The presence of pus and the absorption constitute a factor which contributes to the exhaustion of the patient. Exhaustion hasn't been mentioned in this paper, but I am sure you all realize that as the effect of the burn goes on, with the loss of body fluids, the patient naturally becomes exhausted. That is one of the reasons for frequent blood transfusions. The removal of the coagulum as shown in the movie is the practice that we carry out. Where there is infection we must remove it.

The book that Dr. Luce referred to is very interesting, and when the mention of the brine bath was brought out in the book, it brought to my mind the work of Dr. Blair. He is a strong advocate of the brine bath, and for the old, protracted case, that seems to resist all treatment, the brine bath should always be considered; that is, in the late stages of the burn, where the epithelialization is slow. Where the wound is infected, it is our practice to place the patient in the brine bath.

I wish to take this opportunity to thank Dr. Mock for his remarks. We all owe a great deal to Dr. Davidson, and, as has been mentioned, he stimulated a great many to think of the treatment of burns. It is unfortunate that he should have been taken so early in life.

The time period for the application of tannic acid is, I think, a personal problem. We have cleaned up wounds coming in twenty-four hours after the accident and applied the tannic acid, and we have been successful. I think the burn that comes in, we will say, forty-eight hours old, and which is infected, should not have tannic acid; it would be a great mistake to apply the tannic acid to this type of burn. I think in that particular type of case, Dr. Aldrich's work in the use of gentian violet is the treatment of choice. He told me last night that he had a new dye which he thinks is superior to gentian violet. I would say that the infected case which comes in late should not have tannic acid, because there is infection present and pus will collect under the coagulum adding to the exhaustion of the patient.

The question of the application of home remedies, as mentioned by Dr. Mangurian, is also very interesting, and, if we look back on the ideas of some of the people who have gathered a little information here and there, we find that they have used practical therapeutic measures in combating some of the problems.

In an editorial in the *Journal of the American Medical Association* in the November 17, 1934 issue in the correspondence column, a physician in Pittsburgh referred to a paper that was published in the *Medical Review* in Pittsburgh in 1890. He mentioned the fact that tannic acid was used as a 5 per cent solution by soaking the sponge in the solution and squeezing the solution onto the burned area.

If you go into the history of burns and go back into the work of the Chinese hundreds of years ago where they used tea, you will find that they were thinking of the same problem and trying to arrive at some method of treatment which would lower the mortality. That is our responsibility as physicians, to lower the mortality of any condition we may treat.

I wish to thank the Society for giving me this opportunity to present this interesting subject.

## THE HEART IN RHEUMATIC FEVER*

BY CLIFFORD L. DERICK, M.D.†

BEFORE discussing any phase or manifestation of a disease, it would seem wise to pause and consider the disease itself in its entirety. The question may well be raised as to what Rheumatic Fever is. A definition in other than descriptive terms is impossible at the present time. Dr. Homer F. Swift, who has spent the past fifteen years in an exhaustive study of this disease, has defined Rheumatic Fever as "A disease of undetermined etiology characterized by fever and a toxic state, and by the presence in certain organs of the body of small disseminated focal lesions of a proliferative type. In acute stages there is also an exudation in and about the joints, and sometimes in the pleurae and pericardium. A further characteristic is the tendency for the febrile and arthritic symptoms to disappear when the patient is given large doses of compounds of salicylic or phenyl cinchoninic acids."

Let us study this definition more in detail. First, it is a disease of undetermined etiology characterized by fever and a toxic state. The causative agent of rheumatic fever is still unproved though most workers in this field consider it to be some member of the streptococcus family. Although in its more acute stages it runs a febrile course with leucocytosis, sweating, rapid pulse rate and other manifestations commonly associated with an infection, there most probably is not an invasion of the blood stream by the streptococcus. Existing evidence is more in favor of there being a general tissue hypersensitiveness to the streptococcus similar to what is found with the tubercle bacillus in tuberculosis. Following a tonsillitis or other infection, usually of the upper respiratory tract, due to the streptococcus, toxic products of this organism are carried by the blood stream and cause lesions wherever they may lodge. Support for this theory is obtained by the type and severity of response shown by rheumatic individuals when extremely small amounts of killed streptococci or their products are injected either into the tissues or intravenously. A virus has been claimed as the etiological agent

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by some workers but as yet there is little evidence to substantiate this conception.

Secondly, the definition states "the presence in certain organs of the body of small disseminated focal lesions of a proliferative type". These focal lesions are considered characteristic of rheumatic fever in the same sense as the tubercle is for tuberculosis and the gumma for syphilis. Their nature and composition will be discussed later. These lesions are always in and around blood vessels and are most commonly found in all parts of the heart, the synovia of joints and the subcutaneous tissues where they manifest themselves as subcutaneous nodules. They are found also in the lungs, kidneys, tonsils, serous membranes, and fatty tissue about various abdominal organs. It is most probably true that they occur in any part of the body where there are blood vessels. It is for this reason that nowadays rheumatic fever is considered a general disease and not simply an arthritis with fever. This conception really dates back to 1888 when it was recognized how frequently the heart was involved and left permanently damaged. In other words carditis is as much a part of the disease rheumatic fever as is arthritis. In fact, one may go farther and state that in children, in which age group rheumatic fever is most common, carditis is more frequent than is arthritis. At this age, involvement of only one or no joint may be observed. The converse holds true in adults where arthritis is usually outspoken and carditis occurs with less regularity.

Thirdly, in the acute stages there is also an exudation in and about the joints, and sometimes in the pleurae and pericardium. That this statement holds true for the involved joints has been recognized always and needs no further comment. Until recently, the presence of fever and free fluid in the pleural spaces was considered to be tuberculous in origin. It is now known, however, that in children especially this effusion frequently is part of a rheumatic fever infection. This is proved by the failure to demonstrate tubercle bacilli in the fluid, the failure to have tuberculosis manifest itself elsewhere in the body and, more especially, by having other evidences of rheumatic fever such as arthritis or carditis appear during the course of the illness.

Fourthly, the tendency for the febrile and arthritic symptoms to disappear when the patient is given large doses of compounds of salicylic or phenyl cinchoninic acids. The action of these drugs upon the exudative manifestations of rheumatic fever is almost specific. So much so that many recommend the administration of these drugs as a diagnostic aid. Neither fever nor effusion when due to any other cause, such as tuberculous infection, responds to any comparable degree. The therapeutic use of these drugs will be discussed later.

Thus rheumatic fever as a general disease with protean manifestations is easily visualized. It may have struck some of you as peculiar that up to now I have not used the term acute rheumatic fever. My reason for this is that in most instances rheumatic fever does not run an acute course and terminate with no ill-effects. It is noteworthy that in 1928 the American Heart Association in its publication entitled "Criteria for the Classification and Diagnosis of Heart Disease" omitted the use of the adjective acute.

The disease may run any one of three different courses—

(a) Monocyclic.
(b) Polycyclic.
(c) Continuous.

*(a) Monocyclic*—About twenty-five per cent of all cases have a monocyclic course, that is, after an active attack, evidence of the disease disappears and so far as one can judge, the patient is cured. This is found to occur much more frequently in adults than in children and there is less likelihood of permanent damage to the heart in this group of cases.

*Slide I.** This slide shows the story of an attack of rheumatic fever in two men, one thirty-three and the other forty-seven years of age. In each instance it was the first attack.

It is worth pointing out what observations on these patients are recorded at the Hospital of the Rockefeller Institute in New York City. An attempt is made to fill in the story of joint involvement before admission to the hospital. During the hospital stay observations on temperature, pulse and respiratory rates, white blood cells, conduction time of the heart, condition of joints, administration of drugs, weight, and fluid intake and output are recorded daily or at frequent intervals as shown in these charts.

In the case of this first man, the temperature quickly returned to normal and his joints cleared up. No antirheumatic drugs were used. This patient was kept in the hospital for forty days without further evidence of activity and he has remained free from any recurrence since.

The second patient can hardly be classed as having run a monocyclic course as he had a recurrence of joint activity with a slight elevation of temperature, as shown in the chart, in spite of the continued use of salicylates.

*Slide II. (b) Polycyclic.* It is much more common—over fifty per cent of all cases—to have the disease run a polycyclic course. That is to have all or most evidences of activity clear and then to have the patient go through another attack very similar in every way to the first. These recurring cycles occur with considerable regularity about every twenty to thirty days in the different patients. It is quite usual to find each succeeding attack

*Slides not submitted for publication.

less severe than its immediate predecessor. As time goes on, it would seem as though the patient develops an immunity and in this way overcomes his infection. With each succeeding cycle the likelihood of cardiac involvement with permanent damage becomes greater and is almost certain to have occurred after the third or fourth recurrence.

*Slide III. (c) Continuous.* In this form the patient is never without evidences of activity of the disease. In all the heart is damaged, usually quite seriously. This chart is that of a boy who was studied first at the age of twelve years. From then until his death six years later he spent a good part of the time in the hospital. He was never without evidences of activity of the disease. Some of these evidences were rapid heart action, more or less continuous elevations of temperature and white blood counts and the presence of subcutaneous nodules. The occurrence of new nodules is represented by the solid dots and persistence of old nodules by the circles. His auriculoventricular conduction time was never prolonged to any noteworthy degree. Since he showed few exudative phenomena, drugs were of very little value in this case.

*Slide IV.* This slide shows the course of the disease in a twenty-two year old girl. She had had arthritis for fifteen days before admission to the hospital and precordial pain for four days. As shown by the chart, she ran a very stormy course. The arthritic manifestations responded well to rather large doses of sodium salicylates. She, however, had pericarditis with effusion which cleared slowly after tapping of the pericardial sac. The most noteworthy thing in this patient was the marked delay in the auriculoventricular conduction time. Normally this interval should be under 0.2 seconds, whereas in this patient it was delayed to as much as 0.36 seconds. This delay is evidence of myocardial damage. That this girl had involvement of her entire heart is unquestionable since in addition to the above findings she was left with damaged mitral and aortic valves.

One may ask why all this interest in rheumatic fever. The answer is easy to give, namely, that a large percentage of patients with this disease will be left with hearts which have been damaged to a greater or lesser degree. Since rheumatic fever is not a reportable disease, it is impossible to get statistical evidence as to its frequency. However, it is claimed that patients with rheumatic arthritis make up three to seven per cent of all admissions to large general hospitals in this country and in Europe. These estimates do not include instances of the disease with carditis but without any joint involvement, otherwise the incidence of rheumatic fever would be very much higher. Approaching the problem from a different angle, several matters of interest have been gathered by different workers. Paul, of New Haven, states that among a population of 100 million there are 840,000 with rheumatic heart disease. Thus there are over a million individuals in the United States who have rheumatic heart disease. Another finding of importance is that both rheumatic fever and rheumatic heart disease are much more common in the Northern than in the Southern states. Harrison and Levine, in 1924, collected information from hospitals and practicing physicians in several different cities. Their report, based upon both clinical and autopsy studies, showed both rheumatic fever and rheumatic heart disease to be more than ten times as common in Boston as in Richmond, Va., New Orleans or Oklahoma City. St. Louis and Baltimore occupied intermediate positions in frequency of the diseases as in geographical location. Paul White, in his recent book on heart disease, states that rheumatic heart disease is the most common type of heart disease in New England. His figures are forty per cent of all types of heart disease and—what is of greatest importance—that ninety-three per cent of all cases under twenty years of age are of rheumatic etiology. It is apparent, therefore, why we, who practice medicine here in New England, where rheumatic fever is so prevalent, should be fully aware of its various manifestations.

It was stated previously that the characteristic findings in rheumatic fever are small focal lesions of a proliferative type situated in and about blood vessels. They were first described as occurring in the heart, in 1904, by Aschoff whose name they still bear. Numerous workers since that time have not only confirmed Aschoff's findings but have noted the similarity of lesions occurring elsewhere throughout the body to these in the heart. We, in our studies at the Rockefeller Hospital, were much interested in the nature and occurrence of these lesions. Material for study was obtained by biopsy of subcutaneous nodules and synovia of joints at different stages in their course. In addition, five patients who died moderately early in the course of their disease, came to autopsy. Thus an opportunity was had for a comparative study of the different lesions. Other workers, notably Von Glahn and Pappenheimer, who have a wealth of autopsy material at the Presbyterian Hospital in New York City, have found the same type of lesions about blood vessels in numerous other locations such as lung, perirenal fat, ovarian cyst wall, mesentery, etc. All agree that the typical lesions are made up of collections of large cells with round nuclei and occasional polymorphonuclear leucocytes located always about blood vessels. In addition to the above, the typical Aschoff body in the heart contains numerous characteristic giant cells. During the earlier or more acute stages quite marked edema is often found in and about these lesions. Later when they become

chronic the edema disappears, fibroblasts make their appearance and gradually convert the lesions into areas of scar tissue. The following slides illustrate very well the similarity of the lesions as regards structure and location.

*Slide V.* This slide is of a subcutaneous nodule early in its development. One is at once struck with the intense vascularity and active cell response, which, though general, is most marked in the immediate neighborhood of the vessels. Quite marked edema scattered about the field is noteworthy also.

*Slide VI.* This slide represents synovia of a knee joint during its acute state. Again the vascularity and cellular response are quite apparent.

*Slide VII.* This slide is from the myocardium of a patient who died early in the disease from an overwhelming infection. Here one should note the marked cellular response inside as well as about the vessel. Giant cells, which are found only in the heart lesions, are quite numerous.

*Slide VIII.* This slide, which is from the same patient as the last, shows endocardium and adjacent myocardium. One is at once struck with the marked cellular response in the endocardium as well as in the myocardium.

In many instances the pericardium is involved and shows a similar picture. It is for this reason that one usually speaks of carditis in rheumatic fever rather than attempting to specify the part of the heart involved which the terms endocarditis, myocarditis or pericarditis would suggest. Though as time goes on, evidence that one part was more damaged than the others becomes apparent, it is well to remember that these other parts seldom escape without some damage.

Realizing that the heart is so frequently involved in rheumatic fever, one may ask what the clinical evidences of this involvement are. It should be stated at the outset that we do not intend to discuss the most common end-results in the heart, namely chronic valvular disease and adherent pericardium.

Evidences of cardiac involvement during rheumatic fever can be grouped under several different headings such as—

(1) Symptoms.
(2) Signs.
(3) Laboratory findings.

(1) Symptoms of importance are the following:

(a) Palpitation. When this occurs in a patient at rest or after mild exertion or excitement, it means that the heart is almost surely involved in the disease process. Palpitation occurs with much less provocation in the rheumatic than in the normal heart during the course of any other infection.

(b) Precordial hyperesthesia, tenderness and pain. These symptoms occur in succession or all may be present at the same time. When well marked in the course of rheumatic fever, they usually signify more or less severe heart damage. The exact mechanism of their production, when not due to pericarditis, is not known since it has been shown that direct irritation of the heart muscle does not produce pain. Hitchcock and Swift, after a review of the literature, state that the pain probably originates from lesions in the walls of blood vessels of the heart or in the aorta.

(c) Cough. At times cough may be a troublesome symptom in rheumatic fever. Eliminating the few cases who have the "so-called" irritable cough which has no significance, the remainder fall into three well-recognized groups. First, those where the cough is due to some lesion of the lungs, such as bronchopneumonia which is not uncommon in the early stages of rheumatic fever; secondly, cases of pericarditis with effusion sufficiently abundant to cause pressure on the trachea or large bronchi, and, thirdly, those where there is a marked enlargement of the left auricle which presses upon and irritates the left recurrent laryngeal nerve as it passes around the arch of the aorta. When due to this latter cause, cough is most annoying and very difficult to treat.

(d) Shortness of breath. Shortness of breath in a patient with rheumatic fever practically always is of grave significance. When it occurs only with exercise or excitement, it means that the weakened heart has been overtaxed temporarily, but when it occurs at rest or with the patient lying flat in bed it means serious cardiac damage unless it is due to an extracardiac cause such as pneumonia.

(2) Signs: There are several signs which can be easily observed and which are of much value in estimating whether the heart is involved and to a certain extent the degree of involvement.

(a) Heart rate: A knowledge of the heart rate is very valuable in sizing up the state of the myocardium. A fast rate in the presence of fever is not significant, but a continued fast rate in the absence of fever means a damaged myocardium and one that will need protection from exertion over a prolonged period of time. Again, the rapidity with which a heart returns to its usual rate after slight exercise, such as walking a short distance on the level or climbing a flight of stairs, is a good index as to how much activity that patient can enjoy safely. A normal heart should have slowed to its usual rate within five minutes after moderate exercise, while a damaged heart requires much longer, depending upon the severity of the lesion.

(b) Rhythm: Disturbances of cardiac rhythm are not uncommonly met with in rheumatic fever and when present practically always indicate that the heart is involved. The less serious forms of disturbed rhythm are absence of sinus arrhythmia, which is normally found in children, and the presence of extrasystoles. The more serious arrhythmias are heart block, auricular flutter and auricular fibrillation. These latter always indicate serious myocardial damage frequently with an accompanying pericarditis. The nature of most of these irregularities can be detected at the bedside. This, however, is not always true, for at times the evidence of extrasystoles or some degree of heart block is so similar that differentiation can be made out only by means of an electrocardiogram. They will be discussed later under Laboratory Aids.

(c) Venous engorgement and peripheral edema: When other causes of obstruction to the return flow of blood can be ruled out, the presence of engorgement of the neck veins or edema of the lower extremities signifies a very much weakened and inefficient heart. The presence of these signs always means a much damaged myocardium which frequently is further overloaded by the presence of incompetent valves.

(d) Size of heart: Signs of slight cardiac enlargement are frequently present in the acute stages of rheumatic fever and may have no great significance. On the other hand moderate to marked enlargement does have considerable significance and is due either to an accumulation of fluid in the pericardial sac as part of a pericarditis or to hypertrophy and dilatation of the heart itself which means that there is a severely damaged myocardium and probably endocardium as well.

(e) Heart sounds: Useful information can be obtained by listening repeatedly to the heart in rheumatic fever patients. Things to be listened for are the character and intensity of the sounds, whether there is a reduplication or gallop rhythm, the presence or absence of murmurs and the presence or absence of a friction rub.

Heart sounds are diminished in intensity in the presence of pericardial effusion but more frequently the diminution is due to loss of tone and weakness of the heart muscle when this has been widely involved by the rheumatic process.

Reduplication of sounds or gallop rhythm is observed frequently to come and go during an attack of rheumatic fever with carditis but its cause is unknown unless it means that the muscle of one side of the heart is more damaged and weaker than that of the other side.

The continued presence of murmurs is of definite significance though they of themselves are not given at present so much weight as in previous years. This is due to the fact that murmurs which are easily heard when the heart is rapid may disappear entirely when the rate is slowed and probably when a slight dilatation of one or another chamber of the heart has disappeared. Dr. Henry Christian teaches us not to place too much value as to diagnosis and prognosis of the amount of damage to a valve on any murmur heard during the acute stages of the disease when fever is present. On the other hand murmurs, especially diastolic in time, which are present at a later stage have very definite significance. The valves of the left side of the heart, namely mitral and aortic, are much the most frequently involved.

The presence of a friction rub over the precordium is the most conclusive evidence that the pericardium has been involved. Since these rubs may be of short duration it is important that auscultation be repeated daily, especially in the presence of precordial pain or hyperesthesia which may be the first symptom of pericarditis.

(3) Laboratory evidences of cardiac involvement.

The outstanding laboratory aid in determining whether the heart is involved is the electrocardiograph. Its great usefulness lies in the fact that it will pick up and determine the true nature of lesser degrees of heart block which can be made out in no other way. Since the conduction of the cardiac impulse from auricles to ventricles is intimately associated with the heart muscle any changes observed in the conduction system must mean an involvement of the myocardium. The electrocardiograph is of no aid in determining whether the endocardium or pericardium has been invaded.

Heart block as found in rheumatic fever may be of three different degrees:

(a) Delayed auriculoventricular conduction time.
(b) Dropped beats.
(c) Complete heart block.

*Slide IX.* This slide shows a normal electrocardiogram. Note that the time interval between the P and R waves, or auriculoventricular conduction time, is short—less than 0.2 seconds.

*Slide X.* This slide shows delayed auriculoventricular conduction. It is quite common in rheumatic carditis, as in this instance here, to find the conduction time delayed to much longer than 0.2 seconds without failure of any of the impulses to get through to the ventricles. Since block of this degree gives no evidence that can be detected on physical examination, it must be determined by mechanical means.

*Slide XI.* (b). Dropped beats: These occur when the A-V interval is so lengthened that some of the impulses do not get through to the ventricles. When such an impulse fails to get through, the ventricles are still for a longer

time than usual. These pauses or blocked beats can be observed clinically, but as noted previously the only sure way to differentiate them and extrasystoles is by means of an electrocardiogram.

*Slide XII.* (c). Complete heart block. This is the most marked degree of disturbance of the conduction system when no beats get through from the auricles to the ventricles. In this case the ventricles take on their own independent rhythm. Since the rate when originated in the ventricles is a very much slower one, a change from ninety beats or over to the minute to under fifty should make one suspect complete heart block.

The cause of these alterations in the conduction system is supposed to be and probably is due to the presence of Aschoff bodies in or near the conduction bundle of His. Levy of New York has observed alterations of conduction to parallel the administration of salicylates and believes that when conduction is interfered with there is edema in and about Aschoff bodies situated near this bundle. After administration of salicylates, this edema clears similar to a disappearance of fluid in and about the joints thus leaving the conduction system to function normally.

One final question might be asked and that is as to how frequently the heart is involved in rheumatic fever. The best observations in answer to this question are those of Cohn and Swift who studied a series of cases with daily electrocardiograms. Their findings are summarized on this slide.

*Slide XIII.* They divided their cases of rheumatic fever into three groups as follows: (1) first attack, (2) recurring attacks and (3) cardiac or continuous type.

(1) There were seventeen cases in the first group and of these fifteen showed changes in conduction time and sixteen changes in the form of the ventricular complex. Thus nearly all showed evidence of myocardial involvement. In this same group, six showed definite, ten doubtful and one no evidence of endocardial involvement while three patients developed pericarditis.

(2) In their second group with recurring attacks of rheumatic fever they had twelve cases. Of these ten showed delayed conduction and eleven some alteration in the form of the ventricular complex. All but two cases showed evidence of endocardial involvement and one had pericarditis.

(3) In their third or cardiac group, eight patients were studied. All showed alterations in the form of the ventricular complex and all had evidence of endocarditis while three had pericarditis.

When combined into a composite group one sees that ninety-five per cent of all patients studied showed some evidence of myocardial involvement. Sixty-five per cent showed undoubted evidence of endocardial involvement but this figure is probably too low, as many of the doubtful cases in the first group would likely develop some signs of endocarditis after a period of time. Nineteen per cent of all cases had involvement of the pericardium. Thus one may state that in practically every case of rheumatic fever the heart is involved to a greater or lesser degree. Of course, that does not mean that the damage is permanent in all cases with evidence of conduction changes only. On the other hand the danger is sufficiently great to make one realize that every individual who has an attack of rheumatic fever should be suspected of having some cardiac involvement and that treatment should be so directed as to save the heart as much as possible during the acute stages of the disease.

Prognosis as regards the future of the heart in rheumatic fever is very difficult. One may say that the heart is spared frequently from permanent damage during the first attack, seldom during the second attack and probably never after further attacks. In the cardiac or continuous form, all hearts are damaged to a greater or lesser degree.

### TREATMENT

The ideal treatment would be, of course, the prevention of rheumatic fever, thereby removing the cause of this type of heart disease. As is true of any disease when the cause is unknown, attempts at prevention are more or less empirical. There is much evidence to show that each attack is ushered in by an acute infection usually of the upper respiratory tract. In view of this it was felt that removal of the tonsils and adenoids might prevent the disease. This hope has not been realized as shown by Kaiser working in Rochester, N. Y. After studying large groups of school children both with and without tonsillectomy he has come to the conclusion that the incidence of first attacks of rheumatic fever is slightly less among those who have had their tonsils removed at some previous time but that the removal of tonsils after an attack of rheumatic fever has little or no effect on the incidence of recurrences. Probably the sanest attitude as regards tonsillectomy is not to advise operation unless there is evidence that the tonsils are diseased as shown by recurring attacks of tonsillitis or the persistent presence of enlarged tender submaxillary lymph nodes which drain the tonsillar areas.

In a patient with rheumatic fever the most important form of treatment by far is rest both during and for a considerable time after

the attack. The English school is so convinced of the value of rest to spare the heart that they treat rheumatic fever much as we treat tuberculosis in this country. That is, they have sanatoria or rest homes where the patient can get rest and good nursing care for a prolonged period of time. One who has worked on rheumatic fever over a period of years cannot help but realize how discouraging it is to parents, patients and the physician himself to have to recommend prolonged rest for a child who has no joint pains and who on casual examination appears perfectly normal. It is only after the dangers of serious cardiac damage with their consequent permanent handicap are made clear that it is possible to get the coöperation of either parents or patients. In New York City a group of special teachers are employed who conduct teaching in the homes for those who have persisting infection and are unable to attend school. It is to be hoped that other communities will follow this example.

The injudicious use of drugs such as aspirin, and pyramidon is to be discouraged. These drugs relieve the joint symptoms, which ordinarily bring the patient to the physician, but most definitely do not cure the disease, or, in fact, have any influence in preventing or curing any involvement of the heart. Evidence for this statement is that rheumatic heart disease is more prevalent now than it was before the salicylate era while the incidence of rheumatic fever itself has changed little if at all. Hence, the family bottle of aspirin which can be used whenever there is an ache or a pain is a very serious problem. Too often a child gets up with malaise, with or without joint pain and after an aspirin tablet feels enough better to be allowed to go to school. In these cases it is only when the joints become really acute or, more frequently, when the child has other symptoms such as shortness of breath on exertion that he is brought to the physician. Too late then a true picture of what has been going on is discovered. Aspirin should never be given to children or young adults except with the knowledge and advice of their physicians.

The treatment of the heart after it has started to fail is identical with that for failure from any other cause. In addition to rest and taking as much load off as possible, one may try the use of digitalis. The benefits of this drug during active phases of the disease except in the presence of auricular fibrillation are often disappointing. However a trial is warranted, since one occasionally sees spectacular slowing of the pulse rate and improvement of the circulation.

The question of a change of abode to secure a more suitable climate for patients with rheumatic fever is often raised. This is especially true now that it is recognized that rheumatic fever is uncommon in more mild climates such as are found in Florida, Puerto Rico, etc. Recently two groups of workers, under Coburn in New York City and Jones in Boston, have sent children with rheumatic fever to some southern place for the winter months. While there the children are practically free from upper respiratory infections and their rheumatism is quiescent. However, in most instances the disease has become active again as soon as the children are brought home. Hence if they are to remain free from active disease they must live permanently in the South. This form of treatment is all right as an experiment or for the well-to-do, but is not generally applicable from an economic point of view, chiefly because it would mean moving a large group of the less well-to-do elements of society.

In closing one can only hope that, with the large number of workers who are studying rheumatic fever, its cause will soon be found. When this has happened its prevention and cure can be approached more sanely.

## RECENT DEATH

**COGSWELL** — Samuel J. Cogswell, M.D., aged sixty-one, medical referee for Rockingham County, died on January 18, 1936, after a week's illness. He was stricken with mumps early in the week and pneumonia set in shortly before he died.

Dr. Cogswell was prominent in fraternal affairs of the Knights of Pythias. He was a member of the American Medical Association, the New Hampshire State Medical Society and the Rockingham County Medical Society.

Dr. Cogswell studied at Bowdoin and was graduated from the University of Vermont in 1897. He had been a practicing physician in New Hampshire since 1902.

Dr. Cogswell is survived by his widow and two daughters, Miss Maude Cogswell and Mrs. Marion Harvell.

# CASE RECORDS
## *of the*
# MASSACHUSETTS GENERAL HOSPITAL

ANTE MORTEM AND POST MORTEM RECORDS AS USED IN WEEKLY CLINICAL-PATHOLOGIC EXERCISES

FOUNDED BY RICHARD C. CABOT, M.D.

TRACY B. MALLORY, M.D., *Editor*

## CASE 22071

### PRESENTATION OF CASE

A forty-four year old Italian machinist was admitted complaining of a productive cough.

The patient had been perfectly well until six months before entry at which time he developed a slight head cold followed later by a nonproductive cough. At the end of the second week he was sent to a hospital where he remained until his admission here. During his hospital stay the cough remained constant and became productive of about two cups of yellowish sputum daily. For about five days, three months before admission, he had blood-tinged sputum but at no time was there gross hemoptysis. For one month the sputum had been quite foul. During the first four months of his illness he had frequent night sweats but there were none thereafter. Six weeks before admission he developed sharp pain in the right axilla which was aggravated by cough and inspiration. The pain persisted for four weeks and then subsided completely. His appetite became capricious and his weight decreased from 168 to 128 pounds. He had frequent frontal and occipital headaches which continued for several days at a time. There were occasional sensations of chilliness and warmth. At times coughing would cause him to vomit.

The past history is noncontributory.

Physical examination showed a well-developed, somewhat emaciated, pallid man. The skin was moist and warm. The mucous membranes were pale and there were many carious teeth. The tonsils were large and ragged. The heart was negative. The blood pressure was 110/80. Flatness was elicited in the right upper chest anteriorly down to the level of about the fifth rib. In this region there were crackling râles, bronchial breathing, and pectoriloquy. Posteriorly on the right side there were fine râles, impaired resonance and pectoriloquy down to about the fifth rib. There was clubbing of the fingers. The reflexes were symmetrical but hyperactive.

The temperature was 98.2°, the pulse 120. The respirations were 28.

Examination of the urine showed a specific gravity of 1.006 to 1.010 but was otherwise negative. The blood showed a red cell count of 3,400,000, with a hemoglobin of 55 per cent. The white cell count was 8,700, 74 per cent polymorphonuclears. Repeated sputum examinations were negative for tubercle bacilli. The stools were negative. A Hinton test was negative. Intracutaneous tuberculin gave a positive reaction with 1:1000 dilution but with none of the higher dilutions.

X-ray examination showed a rather homogeneous dullness involving the entire middle lobe and part of the lower border of the right upper lobe. There were two areas of diminished density close to the upper border of the process.

Throughout the patient's hospital stay his temperature varied irregularly between 98° and 102°. It often continued several days without rising above 99°. His pulse remained elevated at 100 or over. On the eleventh day a bronchoscopy showed congestion and thin green foul pus in the right upper bronchus. He continued to expectorate considerable foul greenish sputum which on one occasion was bloody and contained a few small clots. Another x-ray showed a fluid level at the upper edge of the process and extension into the septum between the lower and middle lobes. Examination of the chest showed dullness from the angle of scapula to the tenth rib. Breath sounds were tubular in quality and showed slight diminution in intensity on the right side. Tactile fremitus was not diminished. Amphoric breathing was heard in the region of the right nipple. The heart was neither enlarged nor displaced. A second bronchoscopy showed pus exuding from the right middle lobe bronchus. One month after entry the intercostal bundles between the third and the sixth ribs were excised and one week later the fourth and fifth ribs were resected anteriorly. Four days postoperatively while the surgical dressing was being changed he suddenly began to cough up large amounts of blood. The hemorrhage could not be controlled and he died shortly afterwards.

### DIFFERENTIAL DIAGNOSIS

DR. FREDERICK T. LORD: Will you demonstrate the x-rays, Dr. Hampton?

DR. HAMPTON: This is an oblique view showing dullness on the right side with an area of rarefaction in the upper portion. Here we see a very definite area of rarefaction in the anterior part of the lung. It seems to have a fluid level. Three or four weeks later an anteroposterior view shows dullness in the region of the middle lobe and the lower portion of the upper lobe. This shadow here is middle lobe and the cavity is in the upper lobe.

DR. LORD: There are no masses?

DR. HAMPTON: No masses or appreciable dis-

placement of the mediastinum that I can be sure of. The heart is over a little toward the right. There is nothing here which could be explained by a consolidated middle lobe and a cavity in the upper lobe on the right side.

DR. LORD: The x-ray extends the information in a desirable direction inasmuch as there is no sharply limited shadow but the increased density fades into the neighborhood. The interspaces are no more narrowed than one might expect from an encroaching process.

DR. HAMPTON: That is the posterior portion of the middle lobe, and up above the anterior margin of the lower lobe. The septum is convex upward and anteriorly, indicating that the upper lobe has been reduced in size; but that could be due to partial destruction of the lobe as well as bronchial occlusion.

DR. LORD: The question of a pleural effusion may be raised. The signs are those of consolidation and not of pleural effusion, but encapsulated effusion may, nevertheless, be present. The signs of cavity are ordinarily absent in a disturbance of this sort and it is difficult to explain the presence of amphoric breathing in the region of the right nipple some distance away from the site of the cavity seen in the x-ray.

The symptoms come in sequence, with first a cold six months ago and later cough. We do not know exactly when he had sputum, but not until after the lapse of two weeks. Pain did not come until he had been ill for four and one-half months and the sputum was foul only after five months. Of course abscess is to be thought of, but this evolution of symptoms is unusual. With uncomplicated abscess we expect the complex of symptoms to be complete within a shorter space of time. Abscess is almost constantly peripheral and pain from invasion of the pleura ordinarily comes early in its course. The appearance of individual symptoms in sequence with long intervals between suggests the possibility that the abscess is complicated by something else.

We would like to know the severity of the headache. Cerebral abscess may complicate lung suppuration and the intensity of the headache may have a bearing on this question.

I have already commented on the physical signs, but it is always desirable to consider the possibility of bronchostenosis and atelectasis with which the signs are dullness, diminished or absent breathing, voice, whisper, and tactile fremitus. The signs here are not consistent with bronchial obstruction, the x-ray findings are against it, and we may conclude that here the bronchi, at least the larger bronchi, are probably open.

Clubbing of the fingers may be regarded as consistent with suppuration or malignancy or both. The blood findings with the low color index are consistent with a secondary anemia, but it would be desirable to have a description of the blood smear. The absence of tubercle bacilli on repeated examination of the sputum is against tuberculosis. But it is desirable to have the sputum examined for such other organisms as streptothrix, actinomyces and blastomyces. A positive skin test with tuberculin is to be expected at this age.

Bronchoscopy should always be considered in the presence of localized lung suppuration. It does not seem necessary, however, to do it in all cases as a routine. It is especially indicated when from the history, the physical signs and the x-ray there is a suspicion of the presence of foreign body or malignancy. It may be of assistance in more accurately localizing a suppurative process for surgical intervention. Here it can hardly be said to have localized the process other than to indicate the presence of pus in the right upper and middle lobe bronchus. A difficulty with respect to localization by this means is that the exudate from an abscess may be aspirated into a neighboring or remote bronchus. Bronchoscopy is of little or no value in the treatment of abscess.

Now with respect to the diagnosis, in spite of the unusual evolution of symptoms, it seems reasonable to conclude that he has a lung abscess. There is nothing to suggest tuberculosis other than that the disease must be considered in all pulmonary lesions. The absence in the x-ray films of subapical mottling and the character of the increased density in the involved region are against tuberculosis and we can, I think, dismiss it as unlikely. The unusual evolution of symptoms makes its necessary to consider another possibility. The x-ray findings are not suggestive of malignant disease of the lung. With lung malignancy the increased density tends to be homogeneous with sharply limited margins and here it is mottled with rather ill-defined margins. Nevertheless, I do not think that the possibility of a hidden malignant disease can be excluded.

With respect to the abscess itself, I can go somewhat farther and say that in view of the long duration, the chances are that it consists of multiple though circumscribed areas of suppuration. The hemorrhage probably came from an eroded vessel. There is also the possibility, rather remote, of a complicating cerebral abscess or malignancy.

DR. DONALD S. KING: The thoracic service struggled with this problem. We raised the question that Dr. Lord has raised as to whether the process was lung abscess following a pneumonia process or whether it was an abscess in association with malignancy. Although we did not feel that there was any definite evidence of malignancy, the patient was bronchoscoped in order to rule out such a process. I think that

we have had more so-called postpneumonic abscesses this year than usual, and we assumed that this was a lung abscess following an infectious process.

DR. LORD: There is nothing suggestive of a lobar pneumonia in the history.

DR. KING: No.

DR. FRED: This patient was bronchoscoped twice at an interval of fourteen days. Bronchoscopy was done to help in determining whether there was any new growth or abscess, and if abscess in what lobe it was confined. No new growth, outcropping or foreign body was seen. At the first bronchoscopy a profuse amount of thin foul pus was seen to come from the right upper lobe. The middle lobe at this first bronchoscopy was absolutely clear. The pathology appeared to be confined from the bronchoscopic standpoint to the right upper lobe, but at the second bronchoscopy pus was seen to come from both the right upper and right middle lobes.

DR. J. H. MEANS: I wonder if the long-continued story that Dr. Lord felt was atypical for acute lung abscesses might be due to a succession of abscesses. We see that occasionally. I remember one patient who had x-rays from another hospital showing abscess on one side and when she came to us she had it on the other side. No doubt she had one that healed up and later had another. That might account for this kind of picture. There is a man with postpneumonic abscess on the ward at the present time. His course is not exactly like this but it is somewhat analogous. The question of cancer is interesting and I suppose it occurred to most of us. However, I should like to ask Dr. Mallory if he would not expect an abscess of this sort due to cancer to be peripheral to the cancer and the bronchoscopist really ought to see the cancer if that was the fundamental lesion here.

DR. TRACY B. MALLORY: That is usually the case. I can remember one case, however, where the abscess had involved the cancer and eaten so much of it away that the bronchoscopist was unable to recognize it. We caught it only histologically and did not recognize it in gross.

### CLINICAL DIAGNOSIS

Lung abscess.

### DR. FREDERICK T. LORD'S DIAGNOSES

Lung abscess.
Question of malignant disease.

### ANATOMIC DIAGNOSES

Chronic pulmonary suppuration and fibrosis with necrosis and cavitation.
Bronchiectasis, right upper and middle lobes, slight.
Pulmonary hemorrhage.
Pulmonary emphysema.
Operative wound: First stage thoracoplasty.
Pleuritis, chronic fibrous, bilateral.
Arteriosclerosis, cerebral and aortic, slight.
Pulmonary osteoarthropathy.

### PATHOLOGIC DISCUSSION

DR. MALLORY: In this patient we found a process in the upper and middle lobes on the right which consisted of a very diffuse fibrosis of the entire lobe with multiple cavities all through it but the cavities appeared to be for the most part out in the pulmonary parenchyma, beyond the limits of the bronchi; so that we had to call them multiple abscesses rather than bronchiectases, although there were a few small bronchiectases. The cavities varied from a centimeter in diameter—there were a number as small as that—up to one three centimeters in diameter which was probably the one in which Dr. Hampton demonstrated a fluid level.

From the histologic point of view there is very little to help us in interpreting this case. It seems to me hopeless to trace the origin of the process. Perhaps the most striking thing was the degree of fibrosis in the regions where you would expect lymphatics, as though there had been a definite fibrosis of lymphatics. We see marked fibrosis of lymphatics in other conditions; for instance, in silicosis it is quite constant, but it can occur also in infectious processes. I think I would be a little inclined to Dr. King's suggestion that this might have followed a pneumonia although certainly the history does not give us any characteristic story to suggest that.

DR. LORD: I have been very loath to accept lobar pneumonia, if that is what you were thinking of.

DR. MALLORY: No.

DR. LORD: Our efforts to incriminate lobar pneumonia have largely failed. The early symptom complex with abscess is seldom that of lobar pneumonia. An origin of abscess in bronchopneumonia cannot be denied, and in a restricted sense this is probably true, putrefactive organisms giving rise to bronchopneumonic processes which break down into abscess.

DR. MALLORY: You would regard it as essentially abscess from the start?

DR. LORD: Yes, regarding the tissue changes arising in consequence of a special type of infection and the development of abscess therefrom as an independent affection.

DR. MALLORY: We do not know at all in these cases how often an infectious process which starts with a single organism such as pneumococcus or streptococcus may be complicated by multiple infections later, but we have the analogy of an infarct, for instance, which may develop into an abscess.

The terminal event was a very profuse hemorrhage in the course of which he bled more

into the bronchial tree than he had into the surgical drainage wound. The entire bronchial tree in both lungs was completely plugged with clots of blood and on the left side there was the acute emphysematous dilatation of the alveoli one sees in a drowned man. Death unquestionably was from suffocation. That is a fairly common terminal event in these cases whether or not they are operated on.

## CASE 22072

### Presentation of Case

A forty-five year old white American clerk was admitted complaining of generalized itching of the skin and painful nodules on the fingers.

Twenty-three years ago the patient became ill with painless swollen ankles which were followed by generalized edema. At this time he was admitted to a hospital where he remained for five months, during which period he gradually improved. He was not entirely free from the edema, however, until five months after discharge from the hospital, and it was almost a year before he could return to his work. Following this for about twenty years, except for nocturia of one time, he felt quite well. About two and a half years before entry he began to feel run-down and developed generalized itching and dry skin. He consulted an osteopath who examined his urine and told him that he had a mild diabetes and kidney disease. He was given injections of liver extract for four months "to build up his blood", and also a course of spinal manipulations which relieved his sense of fatigue. The itching was also lessened in intensity. Eleven months prior to admission he again returned to the osteopath for the same reasons he had given previously. He received weekly injections of liver extract thereafter. During this time his nocturia increased three to four times. Two months before coming to the hospital he had burning pain in the middle three fingers of his right hand, and nodules appeared over the skin in this region. Hot soaks relieved the pain but the nodules persisted. Shortly thereafter he developed a tender swelling upon the right elbow which persisted for about a week. He then had successive similar swellings in the region of the right shoulder, left shoulder, left elbow, and ulnar aspect of the left wrist. All these disappeared except the nodule on the wrist which occasionally afforded him some aching pain. Three weeks ago a firm, nontender nodule, about the size of a twenty-five cent piece, appeared on the back of his right elbow. At no time was there any frank joint involvement. His weight had remained constant for three years.

He had scarlet fever at ten years of age.

Physical examination showed a thin pallid man in no acute distress. The patient was ambulatory and cheerful. The skin had a sallow yellow ochre hue. The retinal arterioles were narrowed and shining and there was what appeared to be an organizing hemorrhage in the left retina. The heart was not enlarged. A rough systolic murmur was heard all over the precordium and a prolonged diastolic murmur was audible at the apex. The blood pressure was 165/90. The liver edge was at the costal margin. The prostate was symmetrically enlarged, firm and smooth. The peripheral vessels were firm, nodular and tortuous. There were several cystic swellings about the proximal interphalangeal joints of the right forefinger some of which were translucent and others opaque.

Examination of the urine showed a specific gravity of 1.008, with a trace of albumin and a green reaction to Benedict's solution without precipitate. The sediment contained an occasional white blood cell and a rare red blood cell and granular cast. The concentration test showed a specific gravity fixation between 1.010 and 1.012. The blood showed a red cell count of 3,800,000, with a hemoglobin of 65 per cent. The white cell count was 12,000, 63 per cent polymorphonuclears. There was no stippling and the platelets appeared to be normal. A Hinton test was negative. The nonprotein nitrogen of the blood was 120 milligrams. The carbon dioxide combining power was 37.8 volumes per cent, the uric acid 4.4 milligrams per cent. The chlorides were equivalent to 107 cubic centimeters N/10 sodium chloride. The serum protein was 4.9 grams per cent. A phenolsulphonephthalein injection showed less than 15 per cent of the dye excreted in one hour. A fasting blood calcium was 10.10 milligrams. The phosphorus was 7.92 milligrams and the phosphatase 9.36 units. A stool examination was negative. An electrocardiogram showed normal rhythm with slightly diphasic but essentially upright $T_2$ and $T_3$. $Q_4$ and $T_4$ were negative.

X-ray examination showed masses of homogeneous calcification without trabeculation surrounding the proximal interphalangeal joints of the right second, third and fourth fingers. Similar calcified areas were scattered along the phalanges of these fingers. The cortex of some of the phalanges was thin and moth-eaten in appearance. Both elbows showed similar calcified masses overlying the distal metaphyses of the humeri. The cortex of the radius in the region of the tuberosity and the proximal ends of both humeri showed haziness. There were similar calcified areas about the acromioclavicular joints. These were also present in the soft tissues proximate to the pedal phalanges. The tibiae and fibulae were not remarkable. The

bones of the pelvis showed slight decalcification. The heart contours were at the upper limit of normal and there was a questionable enlargement of the left ventricle. The skull was riddled with small areas of decalcification, one larger area measuring 1.5 by 1 centimeter. In all of the films the large and small vessels showed a marked degree of arteriosclerosis with calcification, some of them definitely of the Mönckeberg type. A pyelogram showed the kidneys to be extremely small. They secreted very little of the dye. The jaws showed absence of lamina dura about most of the tooth sockets and the bone was radiolucent and said to have a ground glass appearance. In some regions there was definite thinning of the bone. Very few caries were seen in the teeth.

The patient continued to be remarkably alert and active for the amount of azotemia he showed. One observer noted uriniferous odor to his breath. At the end of two weeks he developed a severe diarrhea and had several nose bleeds. This continued for about two days. Shortly afterward the serum calcium was found to be 9.13 milligrams, the phosphorus 7.55 and the phosphatase 4.5 Bodansky units. An arterial blood analysis of the acid base components showed an approximate pH of 6.95. The blood showed a red cell count of 2.96, with a hemoglobin of 60 per cent. A few days later under local anesthesia a tibial biopsy was done. Shortly thereafter the patient began complaining of malaise and appeared to be quite apprehensive. Four days postoperatively he was suddenly seized with severe pain between the shoulder blades which later was localized in the left anterior chest and upper abdomen. There was some radiation down the left arm and associated dyspnea and orthopnea. The radial pulse became imperceptible. The blood pressure was 60/50. The heart sounds were poor and a gallop rhythm was heard. There was slight dullness at the left base and a few crackling râles in the same region. The temperature, which had previously been normal, rose to 99.8°. An electrocardiogram showed a left bundle branch block. $T_1$ was inverted; $T_3$ was upright. The P-R interval was .22 seconds. The Q-R-S was .18. The rhythm was slightly irregular, the rate 60. Lead four showed wide excursions and $Q_4$ was nearly absent. $R_4$ was plus 30 and $T_4$ minus 22. His condition remained unchanged and he died five hours later, one month after admission.

## Differential Diagnosis

Dr. Chester S. Keefer*: From this patient's history, the physical findings, and the course of the disease, it would appear that there were two main diagnostic problems. (1) The nature of the renal lesion associated with metastatic calcification and calcinosis. (2) The explanation of the episode that caused the patient's death. I shall discuss this case under these two headings.

*Associate Physician, Thorndike Laboratory of the Boston City Hospital.

When a patient presents the clinical features of renal failure and metastatic calcification, with or without calcinosis, one considers the following conditions:

1. Primary hyperparathyroidism with nephrocalcinosis, or pyelonephritis with urinary calculi.
2. Chronic pyelonephritis or chronic glomerulonephritis with secondary hyperparathyroidism (renal rickets—chronic pyelonephritis with contracted kidney).
3. Calcinosis and chronic nephritis without evidence of parathyroid hyperplasia.
4. Primary bone disease with amyloidosis of the kidney, pyelonephritis or tubular atrophy (multiple myeloma).

I feel reasonably certain that one can exclude the possibility of a multiple myeloma in this case, so that I can take up the discussion of the other conditions at once.

Let us review the salient points in the clinical record. In brief, the patient was a man forty-five years of age who entered the hospital on account of pruritus and painful nodules on the fingers. At the age of ten years he had scarlet fever which may or may not have been accompanied by interstitial and focal glomerulonephritis; however, from the data available, the first indication of a disorder of the kidneys was a generalized edema which lasted for about ten months. He was then twenty-two years of age. Following this experience he remained reasonably well for twenty years, when he developed a feeling of malaise and generalized pruritus with dryness of the skin. Examination of his urine at that time (two and a half years ago) revealed evidences of kidney disease and mild diabetes. There was a temporary remission of his symptoms of fatigue but nocturia increased. Two months before entering the hospital he noticed painful nodules appearing in the skin of the fingers; later there was swelling over the right elbow; then swellings about other joints of his body.

The examination revealed a thin, pale man with a sallow yellow skin, retinal arteriolar sclerosis with an organized hemorrhage in the retina, slight hypertension without cardiac enlargement, a rough systolic murmur over the precordium and a diastolic murmur at the apex. There were cystic changes about the proximal interphalangeal joints of the right forefinger.

The laboratory examinations revealed albuminuria, and only occasional leucocytes, erythrocytes or casts in the sediment. There was a slight secondary anemia, with a moderate leucocytosis. Renal functional studies revealed a loss of concentrating power, nitrogen retention, decreased phenolsulphonephthalein excretion. There was a phosphate retention, a re-

duced $CO_2$ combining power, a low pH, a slight reduction in the serum proteins, a normal blood calcium and an increased blood phosphatase. X-ray examinations revealed calcification in the subcutaneous tissue about the fingers, elbows, acromioclavicular and toe joints, decalcification of the skull, pelvic bones, phalanges, radii and humeri. There was extensive calcification of the blood vessels, and the kidneys were extremely small without x-ray evidence of stones or calcification.

### X-ray Interpretation

Dr. Aubrey O. Hampton: There is a large area of calcification behind the lower end of the humerus which extends to the joint and farther upward than you would expect the joint to extend. The bones are decalcified, and here you can make out subperiosteal bone absorption or bone destruction. This is a very unusual finding in any disease excepting those associated with parathyroid abnormalities and we consider it one of the most characteristic findings of parathyroid abnormality. The skull shows fine areas of increased and diminished density which produce a mealy appearance of the bone with obliteration of blood vessel channels, diploë, and all the normal structures, and with perhaps some thickening of the bone.

This plate of the leg shows extensive calcification in the blood vessels. Furthermore, as has been pointed out by Dr. Schatzki [I do not think he did the original work], this calcium is arranged in concentric circles so that you can deduce from the x-rays that it is in the media of the artery. This plate of the hand shows further deposits of calcification around the joints and here the calcium is definitely outside of the joint in the anterior aspect of the palmar tissue of the fingers. These bones also show decalcification and some subperiosteal absorption. The blood vessels in the wrists and the fingers are also markedly calcified. There are no cysts in any of the bones. The kidney outlines are very small.

### Differential Diagnosis Continued

Dr. Keefer: In brief, then we have a man with a chronic progressive renal failure who finally develops signs of calcinosis and calcification in his blood vessels. This picture fits in very well with a few cases that have been described in the past (Hubbard and Wentworth, Fontana, Penedee). That is to say, patients who have had symptoms of chronic renal disease for varying periods of time (several months to nine years) finally develop metastatic calcification and calcinosis with decalcification of the skeleton. It apparently has been a little more common to observe the same picture *without* calcinosis in the presence of chronic renal disease. Now, in view of the fact that patients with chronic nephritis and metastatic calcification may have enlargement of one or more of the parathyroid glands, the interpretation of the sequence of events in these cases has given rise to lively and interesting discussion. There are undoubtedly cases in which the hyperparathyroidism *precedes* the renal failure and metastatic calcification. These are instances of primary hyperparathyroidism with pyelonephritis and urolithiasis or nephrocalcinosis—so ably defined and described by Albright and his associates as Types 1 and 2.

In addition to these cases, there are instances in which the renal failure precedes the signs of skeleton demineralization and metastatic calcification, and there seems to be little question that in some of these cases, at least, there is secondary hypertrophy of the parathyroid glands and evidences of hyperactivity. From the chronological data in the history and clinical course, I believe that the present case belongs in this group; namely, chronic renal insufficiency with secondary hyperparathyroidism and subsequent metastatic calcification and calcinosis.

I should outline the sequence of events as follows: the development of chronic nephritis with renal insufficiency, the retention of phosphates, enlargement of parathyroid glands with increased activity, demineralization of skeleton due to increased parathyroid activity, chronic acidosis and loss of calcium phosphate in the stools, and finally the precipitation of calcium phosphate and perhaps carbonate in the tissues of the skin and internal organs.

There is an isolated case on record of calcinosis, and calcification in the various blood vessels and tissues associated with scleroderma, in which death resulted from renal failure, without nephrocalcinosis, hypertrophy of the parathyroid glands or demineralization of the skeleton. In view of the fact that this patient did not have scleroderma or calcinosis before the signs of renal failure, it seems fair to exclude this type of case from consideration.

Now, a word regarding the nature of the renal lesion in the present case. There are three types of renal disease with a prolonged course such as was evident in this patient; chronic diffuse glomerular nephritis, chronic pyelonephritis with contracted kidney and congenital cystic kidneys. The latter condition can be excluded at once on account of the absence of attacks of gross hematuria, or of bilateral masses in the flanks, and by the evidence of small kidneys by pyelogram. The history of the onset of the renal disorder with edema suggests glomerular nephritis rather than pyelonephritis, although it is a little unusual, but by no means unknown, for patients with chronic glomerulonephritis to have such a long course, especially when hypertension and endarteritis obliterans are minimal. I should favor a diagnosis of chronic diffuse glomerulonephritis.

There remains for brief discussion the episode

which caused death. There was a sudden onset of thoracic pain, radiating down the arm with the signs of acute heart failure and peripheral vasomotor collapse, the development of a bundle branch block, dullness and râles at the left base of the lung, and death within five hours. This suggests a coronary occlusion, or a pulmonary embolism. In view of the pain and its distribution, the sequence of events and the absence of a definite source for an embolus I should favor a coronary occlusion rather than a pulmonary embolus.

## Clinical Discussion

Dr. Fuller Albright: Dr. Keefer has taken the wind entirely out of my sails. He discussed almost everything I had to say.

There is one interesting point; as you look at this complicated picture with those extraordinary calcified masses around the joints, renal insufficiency, decalcification, and so forth, you wonder if it is one disease. The fact remains that it is, because I know of two other patients who had the same thing; that is long-standing renal disease, calcium deposits around the joints, marked demineralization suggesting hyperparathyroidism, and arteriosclerosis of the media of the arteries. The other cases came to autopsy and did show enlargement of all the parathyroid glands and bone changes typical of osteitis fibrosa cystica, not osteomalacia. The disease, therefore, is analogous to renal rickets. The only difference is that renal rickets occurs in children and there are changes of the growing cartilage in addition. These changes look like rickets by x-ray but turn out not to be rickets under the microscope. The condition is really osteitis fibrosa cystica occurring in growing children.

My diagnosis on this patient before he came to Dr. Mallory's department was the same as Dr. Keefer's and I expected to find four enlarged parathyroid glands.

I would like to say one word about three different types of conditions which may be confused now in everybody's mind. (1) Parathyroid adenoma which leads to hyperparathyroidism, with increased calcium and phosphorus in the urine, and hence to renal disease. That is one clear entity which everyone has firmly in mind. (2) The second entity starts with renal disease and leads to phosphorus retention and compensatory hyperparathyroidism. We do not know how it gets there but you also have decalcification of the skeleton in this syndrome. The end result is different from the first but not very much. The one main difference in the second type is enlargement of all four parathyroid glands instead of one adenoma as in the first type. It would be very easy to differentiate the two if it were not for the fact that there is a third entity. (3) This starts with hyperplasia of all the parathyroids and results in hyperparathyroidism, with increase of calcium and phosphorus in the urine and changes the same as with the first. We have, therefore, two conditions with four enlarged parathyroids and one condition with one enlarged parathyroid. The two with the four enlarged parathyroids fortunately can be differentiated very easily from each other because where the hyperplasia starts as a primary hyperplasia (it is obviously secondary to something else, but primary as regards the kidney) the histology of the parathyroid tissue is quite different from what it is when the hyperplasia is secondary to renal disease.

It might be worthwhile to theorize a little as to what is the sequence of events that leads to the condition we have before us today. In the first place it unquestionably starts as renal disease—it is only with long-standing renal disease over a period of ten or fifteen years that you get the complete picture. The phosphorus retention is probably the next link in the chain. I believe this is the stimulus causing the parathyroid glands to become hyperplastic. Phosphorus retention tends to lead to a low blood calcium and a low blood calcium stimulates the parathyroids to enlarge. That would explain the enlarged parathyroids but would not explain why you get bone disease. Why you get the bone disease, I do not know. There are two possible ways. In the first place it may be caused by an increased amount of hormone going directly to the bone and causing bone disease. This explanation is reasonable if one believes that the hormone acts in bone tissue. That is not my personal belief. The other possibility is that the bone disease is entirely due to the marked acidosis you see with this condition. This patient, with a $CO_2$ combining power of only twenty-eight, had an extreme acidosis which I hope Dr. Talbott will discuss later. He did a complete electrolyte balance on the patient. We know acidosis will cause changes in the bones of the same nature as those seen in hyperparathyroidism.

Dr. John H. Talbott: If our assumption is correct that the first and primary condition was chronic nephritis, I think it interesting that the duration of the disease should be as long as it was. Twenty-two years ago this patient was hospitalized and treated for chronic nephritis. In the intervening years he had been able to carry on his work in a most satisfactory manner. After admission to this hospital we were able to obtain one sample of arterial blood for analysis of the acid base constituents. There is no rule of thumb whereby in a given patient with a disturbance of the electrolyte balance we can predict the concentration of any one constituent. Hence, we consider the following

to be the irreducible minimum number of determinations to be done to give us an accurate picture of the internal environment of a patient. These determinations are carbon dioxide, chloride concentration, sodium and protein concentration and nonprotein nitrogen.

This patient on examination of his arterial blood had a severe acidosis and the carbon dioxide content of the serum was only twenty-eight volumes per cent or approximately twelve milliequivalents, about half the normal. The chloride concentration in spite of the severe nephritis was normal. The sodium concentration of the serum was only 127 milliequivalents, a decrease of about 12 milliequivalents below the normal. Referring again to the carbonate concentration we remember that this was down about 12 milliequivalents, therefore the principal disturbance was a lowered sodium and carbonate concentration with a normal chloride concentration.

The interesting thing in this patient to me, as far as the disturbances of the acid base equilibrium are concerned, is what we would have done had we seen this patient and made these studies ten or fifteen years ago. It is conceivable that if we had found such a disturbance in a mild degree we might have been able to relieve or prevent the acidosis, which I assume to have been responsible for the long train of symptoms leading up to this particular type of hyperparathyroidism. I am interested in this patient as an example of what might be done regarding the preventive aspect of one of the rarer manifestations of the terminal stage of chronic nephritis.

### Clinical Diagnoses

Chronic glomerulonephritis.
Coronary thrombosis?
Dissecting aneurysm?
Rheumatic heart disease?
Aortic regurgitation.

### Dr. Chester S. Keefer's Diagnoses

Chronic glomerulonephritis.
Renal insufficiency.
Hypertrophy of the parathyroid glands (secondary hyperparathyroidism).
Demineralization of the skeleton.
Metastatic calcification with calcinosis.
Calcification of the mitral valve producing the cardiac murmurs.
Coronary occlusion.

### Anatomic Diagnoses

Chronic glomerulonephritis.
Secondary parathyroid hyperplasia, marked.
Rheumatic heart disease with calcification of the mitral and aortic valves and with mitral stenosis and aortic regurgitation.
Coronary occlusion.
Myocardial infarction.
Calcification of the coronary arteries and peripheral vessels.
Arteriosclerosis, marked aortic.
Osteitis fibrosa of the skull.
Calcified nodules of the elbow, fingers and shoulder.
Hydrothorax, bilateral.
Pulmonary edema, bilateral, slight.
Ascites, slight.

### Pathologic Discussion

Dr. Tracy B. Mallory: The autopsy on this man substantiated in practically every detail the clinical diagnosis. We found an extremely atrophic pair of kidneys, weighing only 85 grams. They were granular as well as small, and the cortex was reduced to only 2 mm. in width. Microscopically they show an extreme grade of atrophy, some persistent glomerular scars, and foci of dilated hyperplastic tubules. The pelves are entirely negative and I think it is possible to rule out flatfootedly any question of chronic pyelonephritis. We feel quite sure this is chronic glomerulonephritis.

The next most interesting finding of course concerned the parathyroids themselves. All four of them were very much enlarged and practically popped into view with almost no dissection. The smallest of the glands weighed two grams and the largest nearly five. We estimated the total weight of the four glands as eleven grams. They were interesting in appearance furthermore because they did not look like any parathyroids that we have seen before. On cutting across them they were perfectly homogeneous cream-colored, and so nearly cheesy that I wondered if we might not find necrosis or caseation. They did not have the characteristic orange color that ordinarily enables one to identify parathyroid tissue. On microscopic examination we found a very diffuse hyperplasia with all the cells of the small "chief cell" variety, practically nowhere throughout the four glands have we found any cells with the abundant, highly vacuolated cytoplasm usually described as "water clear" cells. In what we have chosen to call primary hyperplasia with hyperparathyroidism all the cells are of the "water clear" variety so we have here a very sharp histologic difference. It is interesting that with a glycogen stain this patient's glands showed a more intense reaction than any other parathyroid tissue we have examined whereas the primary type of hyperplasia usually shows very little glycogen.

This is a postmortem x-ray of the spine. It shows very prettily the herniation of the intervertebral discs into the bodies of the vertebrae through the softened cortical layer of the vertebral body.

As to the other findings, the vessels throughout the body were of course very extensively calcified. That is particularly true of the larger vessels of the leg which show a typical Mönckeberg type of medial calcification. It was also true of the coronary arteries. They show an extreme grade of calcification with atheromatous deposits and marked narrowing of the lumen. We found in the coronary arteries at autopsy a fresh clot and we were unable to decide whether it was ante- or postmortem. The right auricle of the heart also contained a fresh looking clot which seemed a little too adherent for postmortem clot but again we could not be sure whether it was ante- or postmortem. The microscopic examination, however, clears up the point completely because there are foci of definite early infarction of the myocardium which we were not able to make out grossly; so the clinical diagnosis was more correct in that respect than our gross autopsy findings. The heart showed also, as was predicted, typical rheumatic valvular disease with both aortic and mitral involvement.

The bones showed distinct softening. The skull was thickened, cut very easily, and the diploë was practically indistinguishable from the cortex. Microscopically the changes were indistinguishable from those of true hyperparathyroidism.

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## "DRUG TO END PAIN IN ANGINA REPORTED"

So ran a headline on the first page of the *New York Times* for the last day of 1935. The Professor of Pharmacology at the University of Maryland had read a paper before the American Association for the Advancement of Science at St. Louis. The drug reported was trichlorethylene.

Trichlorethylene is a highly volatile liquid with a strong, sweet smell, first used as a commercial solvent and varnish, and thus found to produce a curious syndrome in some of the workers who handled it. Among its symptoms were vertigo, nausea and anesthesia of the trigeminal nerve. For some years past it has been used for the relief of trigeminal neuralgia, fifteen to twenty drops being inhaled from a handkerchief until the odor has disappeared, and repeated three or four times a day. Too large or frequent dosage has been thought by some to produce disorientation, acute yellow atrophy of the liver, and even ventricular fibrillation with sudden death. Others, however, have denied its toxicity. There is no question that trichlorethylene does produce vertigo and it should therefore be inhaled, like amyl nitrite, in the recumbent position.

Because of this vasodilating effect, it is not surprising that trichlorethylene has been found to relieve "fifteen out of twenty cases", although the number of cases of angina to be relieved by any treatment will depend largely upon the diagnostic criteria used. What is surprising in the *New York Times* account is that "one cubic centimeter in crystalline form, inhaled as snuff, was said to relieve attacks in one second".

The excursions of the daily press into the medical and scientific worlds should not be read too critically. Those of us who are free to come home from a medical meeting and decide that there is nothing of significance among the novelties of the day have a freedom which is denied to the news reporters. So many people having learned to read—they must be furnished with reading material. Medical progress is good reading material, but in the daily press it must be sufficiently sensational to compete with the highly dramatized news of the day—political, criminal and social. Hence the tendency to make a complete symphony out of a perfectly good folk-song.

Trichlorethylene will prove, we hope, a useful drug for the purpose here announced. We only wish that by ending the pain of angina we could solve the many problems that underlie it.

## The Massachusetts Medical Society

### SECTION OF RADIOLOGY AND PHYSIOTHERAPY

THE Section of Radiology and Physiotherapy of the Massachusetts Medical Society will meet at Springfield on Monday, June 8, at 2:30 P.M. There has long been a feeling among the workers in these specialized fields that neither the scope nor the limitations of their work were fully understood by the men in general practice. The program of the Section has therefore been planned to facilitate a better understanding along these lines.

"The Limitations of the Roentgen Method of Diagnosis." Why is the negative Graham-Cole test not worth 100 per cent in excluding gallbladder disease? Why cannot an osteomyelitis be diagnosed in its incipiency? Why are some fractures missed by the most careful technic? These and other questions of the sort will be discussed by Dr. Harvey W. Van Allen of Springfield, well known in his specialty for many years.

"What May the General Practitioner Expect of Physiotherapy?" This subject will be considered by Dr. Claude L. Payzant of Boston, Director of Physical Therapy at Quincy City

Hospital. Some physiotherapeutic methods are overestimated; others are not so well known as they should be. Dr. Payzant will endeavor to evaluate the entire subject with a view to the standpoint of the man in general work.

The subject of Birthmarks is important and interesting to every man who deals with babies. Much progress along this line has been made in recent years; much of it seems not to have reached the rank and file of the profession. The modern treatment of these embarrassing and disfiguring lesions will be discussed by Dr. J. Harper Blaisdell of Boston. Dr. Blaisdell was one of the first workers in this section to apply radium in dermatological work. He is a member of the Massachusetts General Hospital Staff and has occupied several teaching positions.

There will of course be discussion open to all, and it is hoped that many members outside the Section will take part.

## THE ISSUE: WHY DOES MASSACHUSETTS NOT PROTECT ITS CITIZENS?

In the midst of the discussion of House Bill 34 where one observes some lack of restraint, relatively few persons realize what is at stake, or are aware of the issues involved. If one reviews the arguments which have been presented to legislative committees in the past, omitting a multitude of irrelevancies, they seem to run about as follows: There is a "medical trust", which seeking power for itself rather than the welfare of the people, desires to destroy all medical schools which refuse to do its bidding. This trust controls all the state boards of registration in the United States, even the Massachusetts board, and all the medical schools except a few, which few in the spirit of independence and devotion to freedom, "poor but honest", manage to hold out, especially in Massachusetts, the birthplace and home of liberty.

It would doubtless come as a surprise to the various state boards and to the many influential universities in the United States, both state institutions and those which are privately endowed, to learn that a medical trust controls them all; by some malicious, subconscious control it must be.

The second argument is that medical education has become so expensive through the efforts of the medical trust, that the poor boy cannot get an education in the schools which the trust controls, and therefore the "independent" schools must continue to give opportunity for these poor boys. The fact that a comparatively well endowed medical school has available each year about forty thousand dollars for students who need financial aid, while the "independent" school has nothing for such purpose, and yet makes approximately the same annual charge for tuition, is regarded as having nothing to do with the case.

These two arguments alone, though protean in form, are the stock-in-trade of the opponents of the Bill: the right of the school to do as it pleases and the right of the poor boy to get a medical education!

There is a false philosophy implicated here, in the first place in emphasizing rights without qualification. Whose rights are involved, merely those of the school and of the poor boy? And why, merely because the school wishes to please itself, and because the boy is poor?

The true philosophy begins with the consideration of duties which constitute the cause of rights. A medical school conferring degrees by authorization of the state, and by the requirements of the statute participating in the preparation of candidates for licensure, is no longer a purely private institution: it is quasi-public with duties commensurate with its function. Its right to do as it pleases is not unqualified, it is restrained in the interest of the public in accordance with its prescribed function. If its function is perverted by commercial interest, the chief temptation, it becomes the duty of the state to correct this perversion and to prevent degradation. The reason why the commercial interest is the chief temptation is because the state, by requiring the medical degree as a prerequisite for earning a living in the practice of medicine, has placed a commercial value on the degree.

That the "poor" student has a right to medical education no one questions. Yet it is not an absolute right but qualified by several considerations. In the first place he may not want it; perhaps someone else has insisted on his studying medicine. In the second place he may not have adequate intelligence for it; or he may be gifted, but not in the sphere which would make for success in medicine. Or he may not have enough money to pay for it. Whose obligation is it to see that he gets enough money? If there is detected any obligation to give him a medical education, there goes with it an obligation to give him an education worthy of the name, not a mere pretense. The issue is clouded with sophisms, by misrepresentations, by misunderstood facts, duties, obligations, responsibilities.

Yet the issue should be clear to all. It is the duty of the state to protect the citizens in matters affecting their health and against unqualified practitioners there is especial need. Why does the legislature of Massachusetts refuse to give the citizens of this Commonwealth as much protection against unqualified practitioners as the legislatures of other states give their citizens? Here is the issue, and the question is one which will be asked with increasing insistence until the answer is given. It is the duty of the citizens to find the answer.

## THIS WEEK'S ISSUE

CONTAINS articles by the following named authors:

CLARK, RICHARD J. A.B., M.D. Harvard University Medical School 1931. Assistant in Medicine, Massachusetts General Hospital. Member of Medical Staff, Winchester Hospital. Address: 205 Beacon Street, Boston, Mass. Associated with him are

MEANS, JAMES H. A.B., M.D. Harvard University Medical School 1911. Professor of Clinical Medicine, Harvard University Medical School. Address: Massachusetts General Hospital, Boston, Mass. And

SPRAGUE, HOWARD B. A.B., M.D. Harvard University Medical School 1922. Assistant Physician, Massachusetts General Hospital. Visiting Physician, House of the Good Samaritan. Assistant in Medicine, Harvard University Medical School, Courses for Graduates. Address: 270 Commonwealth Avenue, Boston, Mass. Their subject is Total Thyroidectomy for Heart Disease. Experiences with Twenty-One Patients at the Massachusetts General Hospital. Page 277.

JOHNSON, PEER P. A.B., M.D. University of Vermont College of Medicine 1900. F.A.C.S. Chief of Surgical Service, Beverly Hospital. His subject is The Contribution of the Community Hospital to Better Medical Service. Page 295. Address: 163 Cabot Street, Beverly, Mass.

SULLIVAN, ALBERT J. B.S., M.D. Harvard University Medical School 1927. Associate Clinical Professor of Medicine, Yale University School of Medicine. Attending Physician, New Haven Hospital and Dispensary. His subject is Emotion and Diarrhea. Page 299. Address: 303 Whitney Avenue, New Haven, Conn.

PENBERTHY, GROVER C. M.D. University of Michigan Medical School 1910. F.A.C.S. Associate Professor of Surgery, Medical School of Wayne University. Non-Resident Lecturer, University of Michigan Medical School. Director of General Surgery, Children's Hospital of Michigan. Surgeon, Harper Hospital. Associate Surgeon, Herman Kiefer Hospital. Consulting Surgeon, Receiving Hospital. His subject is Treatment of Burns. Page 306. Address: 1551 Woodward Avenue, Detroit, Mich.

DERICK, CLIFFORD L. M.D.C.M. McGill University Medical School 1918. Associate in Medicine, Harvard University Medical School. Senior Associate in Medicine, Peter Bent Brigham Hospital. Non-Resident Consultant in Medicine, Burbank Hospital, Fitchburg, Mass. His subject is The Heart in Rheumatic Fever. Page 310. Address: 412 Beacon Street, Boston, Mass.

## The Massachusetts Medical Society

## SECTION OF OBSTETRICS AND GYNECOLOGY*

C. J. KICKHAM, M.D., *Chairman*, 524 Commonwealth Ave., Boston, Mass.
R. S. TITUS, M.D., *Secretary*, 472 Commonwealth Ave., Boston, Mass.

### ANALGESIA DURING LABOR

Definite progress has been made in the study and use of analgesics during labor within the past two or three years. The work, published in 1934 by Dr. Irving and his associates and dealing with various types of obstetrical analgesia, was very complete in that it gave an excellent comparison of various combinations of drugs which may be used to alleviate pain during labor. Since the publication of this material, there has been a general increase in the use of the barbiturates, chiefly in the form of pentobarbital or nembutal. There has also been much discussion even to the claim that obstetrical analgesia increases maternal mortality. This has not been true at the Boston Lying-in Hospital, since the maternal mortality for 1934 and 1935 has been lower than previously. Practically all the patients were given pentobarbital during labor over this period. It must be remembered that here doctors specializing in obstetrics are ordering the medication and that certain necessary measures for safety are always available.

The purpose of this article is to impress upon the general practitioner certain points to be remembered when administering pentobarbital or nembutal for the purpose of amnesia during labor. In the first place these patients must never be left alone, even for a single second. They are not responsible and may fall out of bed, or in some way injure themselves. Some patients become very restless and measures must be taken to control them. Naturally, therefore, we feel that these drugs should not be used in the home or in any hospital which is not equipped to handle such a case.

The barbiturates, I believe, increase the incidence of vomiting and mucus. To be sure, this does not happen in every case, but when it does occur the general anesthesia given at the end must be administered by a very competent anesthetist. If not, the patient may aspirate mucus or vomitus which may prove disastrous. This is an extremely important point, and when excessive mucus occurs, in spite of atropine, general anesthesia had best be eliminated. The amount of vomiting is often in relation to how recently

*A series of short selected articles by members of the Section is being published weekly.
Comments and questions by subscribers are solicited and will be discussed by members of the Section.

the patient has eaten and if this is considered when ordering the drug it would seem advisable to give it rectally rather than orally. In cases that enter labor vomiting, I believe it best not to give it at all.

As was pointed out in Irving's work, more babies have to be resuscitated than when no analgesia is used. These babies often have a moderate amount of mucus, and the obstetrician must always be prepared to remove the mucus. It can usually be done by the use of a simple catheter. This is very necessary in order that there be a clean airway before the usual methods of resuscitation are tried.

In spite of the occasional presence of such complications, this form of obstetrical analgesia can be used with safety provided the usual precautions are taken. There is no question that the use of these drugs has decreased the unnecessary use of forceps and manual dilatation. The writer believes that practically all the obstetricians familiar with this drug will agree to this.

Purposely, only the more important complications have been mentioned and, for more detailed information, the writer would advise reading the article written by Irving in 1934 and published in *Surgery, Gynecology and Obstetrics.*

## MISCELLANY

### THE APPOINTMENT OF DR. M. J. ROSENAU

Dr. Milton J. Rosenau, who retired last February as professor of preventive medicine and hygiene in the Harvard Medical School and professor of epidemiology in the School of Public Health, has been appointed director of a new Division of Public Health, established as a part of the Medical School of the University of North Carolina. The purpose of organizing the department is to train students to be health officers. The department was founded by the University with the coöperation of the North Carolina State Board of Health.—*Science.*

### MAINE NEWS

At the Thayer Hospital in Waterville a semi-annual review of mortalities has been instituted and adds much to the value of the staff meetings. These mortalities are critically analyzed from the point of view of mistakes in diagnosis, errors of judgment, technical errors, and the diseased condition of the patient. Critical reviews and follow-up are held on all cases which are discharged from the hospital with unsatisfactory results. These frank analyses of cases have stimulated much interest among the medical men in surrounding towns and result in a large and enthusiastic attendance at the Thayer Hospital staff meetings, held fortnightly on the second and fourth Thursdays of each month.

### ANDROSCOGGIN COUNTY MEDICAL ASSOCIATION

At the last meeting of this association it was frankly admitted that doctors in general are not well enough acquainted with the very important and nationally discussed topic of State or Socialized Medicine. It was suggested that in order that the profession become more familiar with the subject and able to discuss it more intelligently, someone who has had the time to study and who knows the proposition from the doctor's point of view should be asked to present it. We were fortunate in securing the coöperation of Professor Brooks Quimby of Bates College who readily agreed with the suggestion.

This permitted us to prepare the following program for our first meeting of 1936, which was held in the Municipal Court Room, City Building, Lewiston, on Thursday, January 16, 1936, at 8:30 P.M. Subject; A formal debate by four members of the Bates Varsity Debating Squad on "Resolved: That the several states should enact legislation providing for a system of complete medical service available to all citizens at public expense."

All members of the profession were cordially invited to attend, and take part in the general discussion after the debate.

R. A. BELIVEAU, M.D., *Secretary.*

### CUMBERLAND COUNTY MEDICAL ASSOCIATION

At the annual meeting of the Cumberland County Medical Association in Portland, Maine, on December 19, 1935, Dr. Shields Warren of Boston read an extremely interesting paper on "Pathology of Malignant Diseases with Relation to Treatment."

### KENNEBEC COUNTY MEDICAL ASSOCIATION

At the annual meeting of the Kennebec County Medical Association held in Augusta on December 19, 1935, Dr. Augustus Riley of Boston read a paper, illustrated with lantern slides, on the subject "Pain in Relation to the Kidneys." This paper was interestedly received and actively discussed.

EDWARD H. RISLEY, M.D.

### THE APPOINTMENT OF SIR FREDERICK HOPKINS

Sir Frederick Gowland Hopkins, British Nobel prize winner in medicine, one of the world's leading biochemists, and a pioneer in the field of vitamin research, has been appointed to the Harvard faculty for the academic year beginning next September. He is now the Sir William Dunn Professor of Biochemistry at the University of Cambridge, and has been Professor of Biochemistry there since 1914.

At Harvard he will deliver a series of three lectures in the Medical School, as the Edward K. Dunham annual lecturer. The Dunham foundation was established in 1923 for the promotion of the medical sciences, through a gift of $50,000 from Mary Dows Dunham, in memory of her husband, a graduate of the Harvard Medical School in 1886. Holders of the lectureships are drawn chiefly from among

the leaders of foreign medical research and are selected by a committee of Harvard departmental chairmen. The foundation was designed to promote understanding between students and investigators here and abroad.

In 1906, Sir Frederick, working with W. Fletcher, laid the foundation of present knowledge of the chemistry of muscular contraction by his researches into lactic acid production in muscle. In the same year he published preliminary reports of experiments involving "pure diets," which proved the existence of essential amino-acids, and of those accessory factors in foods which were afterwards to be known as vitamins. The full results of his vitamin work were published in 1912.

One of his later contributions, made in 1921, was the isolation from living tissues of the sulphur containing dipeptide glutathione, and the proving of its great importance for the oxidations in living cells. He has also done outstanding work in animal pigmentation. He was awarded the Nobel prize in 1929.

## AN HONOR TO DR. KARL BOWMAN

On January 30, 1936, a farewell dinner was given at the Hotel Brunswick to Dr. Karl Bowman, formerly chief medical officer of the Boston Psychopathic Hospital, who has left Boston to take up his new position as Director of the Bellevue Psychopathic Hospital in New York.

About one hundred persons representing his friends and associates attended the dinner.

Dr. C. Macfie Campbell acted as toastmaster. Entertainment in the form of an original dramatic sketch was furnished by some members of the Boston Psychopathic Hospital Staff, including Drs. S. H. Epstein, G. B. Pearson, J. H. DeShon, H. Hirning, R. Schwab, H. C. Solomon, R. H. Guthrie, F. L. Wells and Frances Hannett.

## COMPARISON OF DISEASE INCIDENCE IN CONNECTICUT WITH 1935 AND SEVEN YEAR AVERAGE

MONTH ENDING FEBRUARY 1, 1936

| Diseases | 1936 Week ending Jan. 11 | 1936 Week ending Jan. 18 | 1936 Week ending Jan. 25 | 1936 Week ending Feb. 1 | Average cases reported for week corresponding to Feb. 1 for past seven years | 1935 Week ending Jan. 12 | 1935 Week ending Jan. 19 | 1935 Week ending Jan. 26 | 1935 Week ending Feb. 2 |
|---|---|---|---|---|---|---|---|---|---|
| Chickenpox | 287 | 177 | 145 | 138 | 114 | 317 | 162 | 147 | 166 |
| Conjunctivitis Infectious | 4 | 3 | 5 | 3 | — | — | — | 1 | — |
| Diphtheria | 4 | 5 | 2 | 3 | 18 | 4 | 11 | 3 | 7 |
| Dysentery Bacillary | 1 | — | — | 3 | — | 1 | 3 | 1 | 1 |
| Encephalitis Epidemic | — | — | — | — | — | — | — | — | 1 |
| German Measles | 87 | 93 | 119 | 127 | 11 | 5 | 6 | 7 | 11 |
| Influenza | 1 | 18 | 18 | 3 | 157 | 239 | 96 | 42 | 80 |
| Measles | 87 | 68 | 87 | 71 | 165 | 429 | 529 | 419 | 558 |
| Meningococcus Meningitis | 1 | 1 | 3 | 3 | 1 | — | 1 | — | — |
| Mumps | 116 | 132 | 115 | 92 | 75 | 52 | 47 | 29 | 77 |
| Paratyphoid Fever | — | — | — | — | — | — | — | — | 1 |
| Pneumonia (Broncho) | 56 | 31 | 41 | 24 | 56 | 70 | 56 | 24 | 51 |
| Pneumonia (Lobar) | 81 | 65 | 53 | 44 | 61 | 98 | 73 | 38 | 37 |
| Scarlet Fever | 78 | 59 | 63 | 56 | 83 | 61 | 65 | 46 | 46 |
| Streptococcus Sore Throat | 1 | 4 | 3 | 3 | 2 | — | 9 | 5 | 7 |
| Trachoma | — | — | — | 1 | — | — | — | 1 | — |
| Trichinosis | — | 1 | — | — | — | 1 | 1 | — | — |
| Tuberculosis (Pul.) | 24 | 18 | 16 | 21 | 25 | 32 | 21 | 22 | 24 |
| Tuberculosis (O. F.) | 3 | 1 | — | 2 | 3 | 2 | 2 | 2 | 1 |
| Typhoid Fever | 3 | 1 | — | — | — | 3 | — | 2 | — |
| Undulant Fever | 1 | — | 1 | 3 | — | 1 | 1 | 2 | — |
| Whooping Cough | 78 | 58 | 74 | 49 | 74 | 82 | 94 | 59 | 88 |
| Gonorrhea | 31 | 31 | 35 | 19 | 46 | 25 | 34 | 35 | 57 |
| Syphilis | 47 | 44 | 40 | 55 | 44 | 45 | 46 | 55 | 61 |

Remarks: No cases of Asiatic cholera, glanders, plague or yellow fever during the past seven years.

## MASSACHUSETTS DEPARTMENT OF PUBLIC HEALTH

JANUARY, 1936

DIPHTHERIA CASES REPORTED FROM CITIES AND TOWNS OVER 10,000 POPULATION

How Does *Your* Community Stand?

| City or Town | 1935 Population* | 1929 | 1930 | 1931 | 1932 | 1933 | 1934 | 1935 | 1935 Case Rate† |
|---|---|---|---|---|---|---|---|---|---|
| Massachusetts | 4,350,910 | 4,255 | 3,322 | 2,381 | 1,811 | 1,041 | 629 | 396 | 9.1 |
| Chicopee | 41,952 | 40 | 21 | 20 | 8 | 3 | 7 | 32 | 76.3 |
| Lowell | 100,114 | 65 | 21 | 26 | 38 | 71 | 76 | 50 | 49.9 |
| Peabody | 22,082 | 21 | 24 | 57‡ | 19 | 12 | 8 | 10 | 45.3 |
| Fairhaven | 11,005 | 13 | 15 | 11 | 11 | 5 | 1 | 3 | 27.3 |
| Chelsea | 42,673 | 55 | 56 | 42 | 44 | 16 | 14 | 11 | 25.8 |
| Fall River | 117,414 | 129 | 117 | 100 | 41 | 34 | 29 | 28 | 23.8 |
| New Bedford | 110,022 | 228 | 160 | 118 | 78 | 73 | 27 | 24 | 21.8 |
| Danvers | 13,884 | 20 | 18 | 4 | 5 | 5§ | 2 | 3 | 21.6 |
| Somerville | 100,773 | 124 | 179 | 119 | 64 | 63 | 54 | 21 | 20.8 |
| Revere | 35,319 | 51 | 29 | 38 | 9 | 11 | 15 | 7 | 19.8 |
| Taunton | 37,431 | 3 | 5 | 38 | 14 | 16 | 3 | 7 | 18.7 |
| Adams | 12,858 | 10 | 17 | 11 | 6 | 1 | 0 | 2 | 15.6 |
| Natick | 14,394 | 2 | 3 | 2 | 0 | 0 | 1 | 2 | 13.9 |
| Boston | 817,713 | 1,104 | 862 | 701 | 540 | 207 | 114 | 113 | 13.8 |
| Southbridge | 15,786 | 9 | 2 | 0 | 5 | 0 | 3 | 2 | 12.7 |
| Winthrop | 17,001 | 7 | 1 | 4 | 3 | 4 | 1 | 2 | 11.8 |
| Northbridge | 10,577 | 35 | 14 | 1 | 1 | 2 | 1 | 1 | 9.5 |
| Swampscott | 10,480 | 11 | 6 | 5 | 5 | 1 | 0 | 1 | 9.5 |
| Athol | 10,751 | 1 | 1 | 0 | 16 | 5 | 2 | 1 | 9.3 |
| Framingham | 22,651 | 6 | 3 | 0 | 2 | 0 | 4 | 2 | 8.8 |
| Everett | 47,228 | 106 | 63 | 103 | 47 | 15 | 4 | 4 | 8.5 |
| Watertown | 35,827 | 35 | 27 | 23 | 13 | 9 | 4 | 3 | 8.4 |
| Gloucester | 24,164 | 14 | 2 | 3 | 1 | 3 | 6 | 2 | 8.3 |
| Webster | 13,837 | 13 | 1 | 0 | 2 | 0 | 0 | 1 | 7.2 |
| Wakefield | 16,494 | 29 | 10 | 2 | 2 | 5 | 4 | 1 | 6.1 |
| Arlington | 38,539 | 18 | 23 | 8 | 19 | 9 | 10 | 2 | 5.2 |
| Woburn | 19,695 | 33 | 9 | 9 | 7 | 6 | 2 | 1 | 5.1 |
| Waltham | 40,557 | 13 | 4 | 4 | 15 | 4 | 3 | 2 | 4.9 |
| Medford | 61,444 | 87 | 47 | 64 | 39 | 41 | 20 | 3 | 4.9 |
| Worcester | 190,471 | 125 | 213 | 117 | 102 | 54 | 23 | 9 | 4.7 |
| Weymouth | 21,748 | 12 | 5 | 8 | 7 | 14 | 2 | 1 | 4.6 |
| Leominster | 21,894 | 2 | 3 | 0 | 13 | 0 | 0 | 1 | 4.6 |
| North Adams | 22,085 | 1 | 2 | 1 | 0 | 3 | 0 | 1 | 4.5 |
| Melrose | 24,256 | 10 | 10 | 8 | 14 | 0 | 2 | 1 | 4.1 |
| Haverhill | 49,516 | 57 | 72 | 6 | 13 | 12 | 3 | 2 | 4.0 |
| Brookline | 50,319 | 5 | 16 | 9 | 7 | 1 | 1 | 2 | 4.0 |
| Belmont | 24,831 | 11 | 6 | 11 | 6 | 2 | 3 | 1 | 4.0 |
| Beverly | 25,871 | 18 | 44 | 7 | 3 | 1 | 6 | 1 | 3.9 |
| Malden | 57,277 | 91 | 24 | 62 | 22 | 11 | 12 | 2 | 3.5 |
| Brockton | 62,407 | 25 | 23 | 48 | 11 | 15 | 1 | 2 | 3.2 |
| Salem | 43,472 | 220 | 133 | 60 | 52 | 18 | 5 | 1 | 2.3 |
| Pittsfield | 47,516 | 54 | 8 | 2 | 2 | 2 | 3 | 1 | 2.1 |
| Cambridge | 118,075 | 144 | 91 | 53 | 69 | 50 | 14 | 2 | 1.7 |
| Newton | 66,144 | 8 | 3 | 1 | 6 | 13 | 2 | 1 | 1.5 |
| Quincy | 76,909 | 17 | 7 | 10 | 26 | 25 | 13 | 1 | 1.3 |
| Springfield | 149,642 | 268 | 133 | 39 | 17 | 9 | 3 | 2 | 1.3 |
| Lawrence | 86,785 | 31 | 16 | 13 | 3 | 3 | 4 | 1 | 1.2 |
| Lynn | 100,909 | 195 | 191 | 106 | 57 | 28 | 32 | 0 | 0.0 |
| Holyoke | 56,139 | 36 | 5 | 3 | 2 | 3 | 0 | 0 | 0.0 |
| Fitchburg | 41,700 | 64 | 16 | 10 | 11 | 2 | 3 | 0 | 0.0 |

MASSACHUSETTS DEPARTMENT OF PUBLIC HEALTH *(Concluded)*

| City or Town | 1935 Population* | 1929 | 1930 | 1931 | 1932 | 1933 | 1934 | 1935 | 1935 Case Rate† |
|---|---|---|---|---|---|---|---|---|---|
| Northampton | 24,525 | 12 | 45‖ | 4 | 2 | 2¶ | 0 | 0 | 0.0 |
| Attleboro | 21,835 | 23 | 6 | 7 | 10 | 6 | 3 | 0 | 0.0 |
| Methuen | 21,073 | 4 | 4 | 2 | 2 | 0 | 1 | 0 | 0.0 |
| Gardner | 20,397 | 2 | 3 | 1 | 0 | 1 | 0 | 0 | 0.0 |
| Westfield | 18,788 | 10 | 7 | 2 | 0 | 0 | 0 | 0 | 0.0 |
| Milton | 18,147 | 0 | 4 | 3 | 3 | 2 | 1 | 0 | 0.0 |
| Braintree | 17,122 | 7 | 5 | 4 | 2 | 1 | 2 | 0 | 0.0 |
| West Springfield | 17,118 | 10 | 6 | 0 | 1 | 3 | 2 | 0 | 0.0 |
| Greenfield | 15,903 | 9 | 5 | 4 | 2 | 0 | 0 | 0 | 0.0 |
| Marlboro | 15,781 | 6 | 2 | 4 | 13 | 1 | 0 | 0 | 0.0 |
| Norwood | 15,574 | 3 | 5 | 2 | 2 | 0 | 0 | 0 | 0.0 |
| Dedham | 15,371 | 2 | 3 | 1 | 0 | 3 | 0 | 0 | 0.0 |
| Saugus | 15,076 | 7 | 6 | 6 | 1 | 1 | 0 | 0 | 0.0 |
| Milford | 15,008 | 10 | 1 | 1 | 3 | 0 | 0 | 0 | 0.0 |
| Newburyport | 14,815 | 57 | 84 | 13 | 14 | 1 | 0 | 0 | 0.0 |
| Wellesley | 13,376 | 2 | 1 | 0 | 10 | 2 | 2 | 0 | 0.0 |
| Winchester | 13,371 | 6 | 7 | 18 | 5 | 0 | 0 | 0 | 0.0 |
| Plymouth | 13,183 | 3 | 11 | 9 | 3 | 2 | 1 | 0 | 0.0 |
| Clinton | 12,373 | 1 | 0 | 1 | 1 | 1 | 0 | 0 | 0.0 |
| Stoneham | 10,841 | 14 | 10 | 2 | 1 | 0 | 0 | 0 | 0.0 |
| Needham | 11,828 | 3 | 6 | 1 | 3 | 3 | 2 | 0 | 0.0 |
| Lexington | 10,813 | 1 | 7 | 3 | 10 | 1 | 2 | 0 | 0.0 |
| Reading | 10,703 | 2 | 17 | 1 | 1 | 1 | 0 | 0 | 0.0 |
| Amesbury | 10,514 | 15 | 10 | 11 | 1 | 1 | 0 | 0 | 0.0 |
| Easthampton | 10,486 | 10 | 2 | 0 | 0 | 0 | 0 | 0 | 0.0 |
| North Attleboro | 10,202 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0.0 |

*Population—State census as of January 1, 1935.
†Case Rate per 100,000 population.
‡Included 25 cases which occurred in a short outbreak in a single school.
§All cases in State Hospital.
‖Included 39 cases in State Hospital.
¶One case in State Hospital.

## MORBIDITY AND ECONOMIC PROBLEMS OF CITIZENS OF BOSTON

Unemployed persons as a group are not so healthy as the employed, according to a Harvard study of the entire population of Boston over a period of six months.

The unemployed, that is, the group seeking work but unable to find it, have proportionately more illness than the part-time or full-time employed. But the group with the greatest proportion of sickness is the not-seeking-employment class, which includes the young, the housewives, the aged, and the chronic sick.

The unemployed have more serious illnesses than the employed, the study showed. The unemployed also receive proportionately more medical care, free or paid, than any other group of the city's population. The temporary and part-time employed have the largest number of cases of illness with no medical care.

The unemployed form the majority of free out-patient and in-patient hospital cases. About one out of every two cases of illness in the city has paid medical care, about one of every three receives free treatment, and one out of six has no outside medical treatment.

Private doctors care for about half of the city's sick. Free private-doctor cases form only a small percentage of the total private-doctor cases. Private doctors care for more patients than do the hospitals. About one of every two cases of sickness is treated by a private doctor, and one of three is treated in hospitals.

In making the survey, illness was considered as any sickness which incapacitates a person for at least two days. Figures for the study were taken from the unemployment census survey for Massachusetts, made by the CWA, showing the illnesses of 769,504 persons in Greater Boston during the last six months of 1933. The study was made by Carle C. Zimmerman, Associate Professor of Sociology; Carl R. Doering, Assistant Professor of Vital Statistics, and Edwin B. Wilson, Professor of Vital Statistics, all of Harvard, and is part of a survey of unemployment conditions in Massachusetts conducted by various Harvard faculty members for the State Department of Public Welfare. Dr. Carl C. Seltzer, of Cambridge, assisted in the study. The summary of results in the official report is as follows:

SIZE AND DISTRIBUTION OF BOSTON SICK POPULATION

"In the City of Boston over a period covering the last six months of 1933, 68,047 individuals or 8.8 per cent of the total population reported one or more cases of illness.

"In proportion to the size of their populations, South Boston, Roxbury, North End, and West Roxbury had more cases of illness than the city average.

"In proportion to the size of their populations, Back Bay, Charlestown, Jamaica Plain, West End, and Dorchester South had fewer sick persons than the city average.

"A total of 68,047 sick individuals reported 76,073 cases of illness for the six-months' period. This averages 1.1 cases of illness per person. There are no district variations for the city in this respect.

"The greatest numbers of sick persons were found in Roxbury and Dorchester North, and the least number in Back Bay, West End, Charlestown, North End, and Hyde Park.

ILLNESS AND EMPLOYMENT STATUS

"There is unquestionably a strong association between employment status and frequency of illness: the proportion of illness increases as the degree of employment decreases; and, conversely, as the degree of employment increases there is a corresponding decrease in frequency of illness.

"In comparison with the other employment groups, the unemployed have proportionately more sickness (9.3 per cent) than the temporary and part-time employed (6.2 per cent), and the temporary and part-time employed more sickness than the full-time employed (3.6 per cent). However, the not-seeking-employment class (which includes the young, the housewives, the aged, and the chronic sick) shows more illness than any of the other groups (11.5 per cent).

"If the employables alone are considered, the unemployed have a much larger representation in the sick population (45.0 per cent) than in the total population (26.2 per cent). The temporary and part-time employed show but a slightly increased representation in the sick population (13.0 per cent) than in the total population (11.3 per cent), and the full-time employed display a substantial decrease in their proportionate representation in the sick population (42.0 per cent) than in the total city population (62.5 per cent).

"High proportions of unemployed sick do not necessarily occur in districts with more than their share of unemployed, nor do districts with more than their share of unemployed have more than their share of illness. Apparently there is no consistent correlation between unemployment and high frequency of illness for all the city districts.

"Districts with more than their share of full-time employed have less than their proportionate share of illness (Back Bay, Brighton, Dorchester South, Jamaica Plain, South End, West End, single exception is West Roxbury).

"North End and Roxbury are the only districts which have more than the city average in number of sick persons and also in the number of persons both sick and unemployed.

"Generally, it was found that a low percentage of illness was not necessarily accompanied by a low percentage of unemployed, and conversely, a high percentage of illness was not consistently reported in districts reporting a high percentage of unemployed persons.

"Unemployment seems to go more with economic status of district than with frequency of illness, for in the economically 'better' districts a high percentage of sick was accompanied by a low percentage of unemployment (Hyde Park and West Roxbury), and a low percentage of sick persons was found associated with a low percentage of unemployed (Back Bay, Brighton, Dorchester South, and Jamaica Plain).

"Conversely, in the economically 'poorer' districts a high percentage of sick was accompanied by a high percentage of unemployed (North End, Roxbury, South Boston), and a low percentage of sick was accompanied by a high percentage of unemployed (Charlestown, South Boston, South End, and West End).

"On the basis of the size of the district population, Roxbury and North End show more than their proportionate share of persons both sick and unemployed. Conversely, Charlestown, East Boston, and Dorchester South have considerably less than their proportionate shares of persons both sick and unemployed.

"According to the size of the district population, Roxbury, West Roxbury, Brighton, and Back Bay possess more than their expected number of persons both sick and temporary and part-time employed; the districts with less than the expected number under this classification are East Boston, Charlestown, Jamaica Plain, and Hyde Park.

"According to the size of the district populations, we find West Roxbury, South Boston, Brighton, and Roxbury with a proportionate excess of persons both sick and full-time employed; Back Bay, East Boston, South End, North End, West End, and Charlestown have considerably less than their expected number of persons both sick and full-time employed.

"Again, on the basis of the size of the district populations, South Boston, Roxbury, South End, and North End have more persons sick and not seeking employment, while Back Bay, Jamaica Plain, Charlestown, Dorchester South, and West End show less than the expected number of persons both sick and not seeking employment.

ILLNESS, EMPLOYMENT, AND TYPE OF MEDICAL CARE

No Medical Care Cases:

"Out of 76,073 cases of illness in the city, 12,201 or 16.0 per cent received no medical care whatsoever.

"The districts with the largest proportion of illnesses with no medical care are South Boston, West End, Roxbury, and Back Bay. Those with the smallest proportion of illness with no medical care

are North End, Jamaica Plain, Brighton, and Charlestown.

"Lack of employment and high proportion of illnesses with no medical care do not necessarily go together, for, persons both sick and unemployed have less cases of illness with no medical care than the persons sick and full-time employed, part-time employed, and not seeking employment. Persons both sick and temporary and part-time employed have the largest number of cases of illness with no medical care.

"Under the family employment classification, the lowest proportion of illnesses with no medical care is found in families with one or more full-time workers, and the highest in families with one or more part-time workers.

Out-Patient Cases:

"Out-patient clinic cases amount to 15.6 per cent of the total number of illnesses in the city for the six-months' period.

"Paid out-patient cases form only a very negligible proportion of the total cases of sickness in the city, the free out-patient cases constituting by far the major part of the total out-patient cases.

"The districts with the largest share of their sicknesses receiving treatment in the out-patient clinics are South End, North End, West End, and Roxbury. The districts presenting the smallest proportion of out-patient cases are West Roxbury, Brighton, Hyde Park, and Jamaica Plain.

"In every single district, as well as for the city as a whole, there is a very strong association between unemployment and high frequency of free out-patient cases. Wherever we find excessive proportions of unemployed we also find excessive frequencies of free out-patient cases.

"This same correlation is also present when the data are sorted on the family employment basis.

"In virtually all the employment status groups, South End, North End, West End, and Roxbury have the largest proportion of free out-patient cases, and West Roxbury, Brighton, Hyde Park, and Jamaica Plain the smallest.

In-Patient Cases:

"In-patient hospital cases in the city of Boston numbered 15,429 or 20.3 per cent of the total cases of sickness reported.

"The distribution of the frequencies of in-patient cases according to city districts is remarkably uniform, except when the cases are sorted on the individual and family employment status and on the paid and free basis.

"The proportion of paid to free in-patient cases varies directly with the employment status of the individual. With an increase in unemployment there is a proportional increase in the size of the free in-patient class and a corresponding decrease in the paid in-patient group. The family employment data reveal this same clear-cut correlation between degree of employment and frequency of free and paid in-patient cases.

"Among the sick and unemployed there are fewer free in-patient cases than the paid type in Back Bay, Brighton, Dorchester South, and West Roxbury; and more free than paid in-patient cases in Charlestown, Dorchester North, East Boston, North End, Roxbury, South Boston, South End, and West End.

"Among the sick and full-time employed, Back Bay, Brighton, Dorchester South, Hyde Park, Jamaica Plain and West Roxbury, all show a much larger disproportion of paid to free in-patient cases than any of the other districts.

Private-Doctor Cases:

"Private-doctor cases amount to 48.1 per cent or about one-half of the total number of illnesses reported for the city of Boston in the six-months' period of 1933.

"West Roxbury, Brighton, Jamaica Plain, Hyde Park, Charlestown, and Dorchester South give higher proportions of private-doctor cases than the city average, while South End, West End, Roxbury, South Boston show smaller proportions of private-doctor cases than the city average.

"Free private-doctor cases form only a small percentage of the total private-doctor cases in the city, except in North End and Charlestown where free treatment by private doctors is not an unusual occurrence.

"There is a strong correlation between the number of free private-doctor cases and increased frequency of unemployment.

"In all the individual and family employment groups, the highest proportions of free private-doctor cases are to be found in the North End, Charlestown, East Boston, South Boston, and South End.

"Paid private-doctor cases form by far the largest proportion of illnesses, and its association with the various degrees of employment is very sharply defined. Paid private-doctor cases make up one-fourth of the unemployed sick cases, one-third of the temporary and part-time employed sick and one-half of the full-time employed sick.

"According to districts, paid private-doctor cases on both the individual and family employment basis are highest in West Roxbury, Brighton, Jamaica Plain, Back Bay, Hyde Park, and Dorchester South.

### PAID VERSUS FREE MEDICAL CARE

"About one out of every two cases of illness in the city has paid medical care, about one out of every three receives free medical treatment, and about one out of every six uses no outside medical assistance whatsoever.

"The burden of medical treatment is borne more by the private-doctor than by the hospitals, for the private-doctor cases amount to 48.1 per cent of the total cases of illness in the city and the hospital cases are but 35.9 per cent of the total city illnesses. Therefore, about one of every two cases of illness is treated by a private doctor, one out of every three treated in hospitals, and about one of every six cases of illness has no treatment whatsoever.

"Out of the total private-doctor cases 86.8 per cent

are paid cases and 13.2 per cent are free cases. With respect to the hospital cases only 30.6 per cent were paid cases and 69.4 per cent were free cases.

"The districts with the largest percentage of illness with paid medical care are Brighton, West Roxbury, Jamaica Plain, Dorchester South, Hyde Park, and Back Bay.

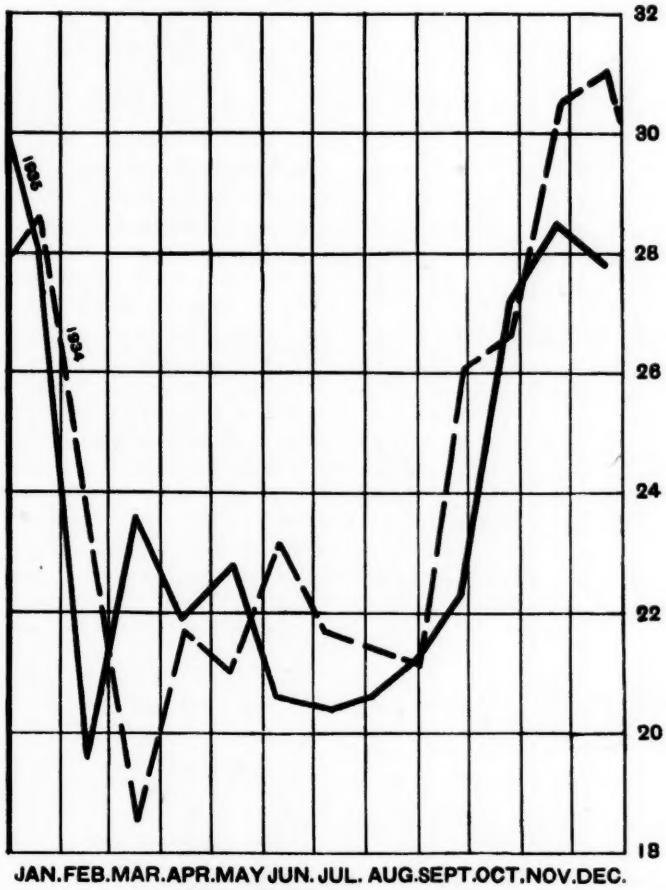
"The districts with the lowest proportions of paid cases are to be found in the South End, West End, South Boston, North End, Roxbury, and East Boston.

"The distribution of free cases, according to districts, reverses the results of the paid cases, for those districts with the highest percentage of paid cases are the same districts with the lowest percentage of free cases, and, conversely, the districts with the lowest proportion of paid cases are the same districts that present the highest proportion of free cases.

"There is a very high correlation between the frequency of paid and free cases, and the degree of employment, individual and family. As we pass from the unemployed to the full-time employed state the frequency of paid cases shows a marked increase, and the frequency of free cases a decided decrease."

## SUMMARY OF MORTALITY FROM AUTOMOBILE ACCIDENTS

DEATH RATES FROM AUTOMOBILE ACCIDENTS BASED ON DEATHS REPORTED FROM 86 LARGE CITIES FOR FOUR-WEEK PERIODS: 1935 AND 1934




Death Rate per 100,000 Estimated Population.

—*Bureau of the Census.*

# CORRESPONDENCE

## PSEUDO MEDICAL HOCUS POCUS

January 27, 1936.

Editor, *New England Journal of Medicine,*

A colleague of mine, being somewhat bewildered, referred the following letter to me for interpretation. It concerns a young patient of his with a rash who was somehow referred to the so-called Research Department of a suburban hospital. The contents of the letter were so remarkable that I thought they might be worthy of publication. With omission of names and with my own parenthetical observations, the communication reads exactly as follows:

"Miss H. C. who is under your care was referred to this laboratory for tests. Miss C. suffers from a slight dermatitis. She is slightly underweight for her age and height. Her bloodpressure is maybe somewhat elevated for her age.

"Blood Morphology: Redcount is normal. The leucocyte count is slightly above normal (sic). There is a lymphocytosis of 66%, and the neutrophils are diminished with an elevation of the young forms to a multiple index of 3.2 (!!). The hemoglobin is a low normal (sic).

"Blood Chemistry: The cellvolume is below normal. The plasma sugar is above the normal for her age (!). The N.P.N. is elevated (sic). The uric acid is an high normal (sic). The plasma NaCl is very much elevated. The inorganic phosphorus is high (sic). The coagulation time is a prolonged normal (sic!!).

"Urines: The patient shows a high specific gravity in all three specimens. There is no alkaline tide in the morning. The specimen after meals show some sugar. All specimen show urobilinogen with an increase toward the evening.

"These tests show that Miss C. is a pituitary type with a dysfunction of the mineral-metabolizing hormone and of the carbohydrate metabolizing hormone. (God help the poor already overburdened pituitary!) This anterior pituitary dysfunction is the cause of her dermatitis (!!), of her low resistance and would in later life be the cause of grave disturbances of the pancreas and the ovarial function. (P-l-lease, doctor!)

"We suggest respectfully to submit Miss C. to a Tuberculin test and to have her chest examined to rule out TBC, since the high phosphorus level in the blood may have some bearing on it.

"We suggest a dietary regime to correct the faulty metabolism and to increase the resistance to infections. The diet must contain large amounts of C.H., iron, calcium and must be especially rich in all vitamines. (Ah!) The fat- and saltcontent of the diet must be severely restricted. As a perfect supply of iron and vitamines we suggest Irradol-A Parke, Davis and Co.

"A dietchart is included.

"We are glad to coöperate with you in question which may arrise (sic) and to supply you with all information we may possess."

The associated laboratory data given with the letter are as follows:

Miss H. C.

| | |
|---|---|
| Age | 8.8 yrs. |
| Wt. | 77 lbs. |
| Bl. Pr. | 112-68 |

Blood Morphology

| | | | |
|---|---|---|---|
| Hemoglobin | 85% | Lymphocytes | 66% (Large 2, small 64) |
| RBC | 5,100,000 | Neutrophils | 30% (seg. 25, stab 5) |
| WBC | 9,600 | Endothelials | 3% |
| Color Index | 0.83 | Eosinophils | 1% |
| | | Basophils | 0 |
| | | Multiple Index | 3.2 |

Blood Chemistry

| | | | |
|---|---|---|---|
| Cells | 39% | Uric acid | 3.1 mg. % |
| Plasma | 61% | Plasma NaCl | 644 mg. % |
| Sugar | 106 mg. % | Inorganic P | 4.2 mg. % |
| N. P. N. | 30 mg. % | Coagulation time | 6 min. |

Urine Chemistry
(morning spec.)

| | | | |
|---|---|---|---|
| Spec. Grav. | 1.023 | Albumen | none |
| Reaction | sl. acid | Bilirubin | none |
| Sugar | none | Urobilinogen | ++ |
| Acetone | none | Indican | ++ |

Sediment: Small amount. Some RBC. 5 leucocytes per HPF, some epithelial cells, few cocci and bacteria, no crystals, no casts.

| | After breakfast | After dinner |
|---|---|---|
| Spec. Grav. | 1.027 | 1.034 |
| Reaction | Acid | sl. acid |
| Sugar | Reduction | trace |
| Urobilinogen | + | +++ |

I don't think the letter needs much comment. With the laboratory data it is composed of about as much pseudo medical hocus pocus as one could possibly gather together in two pages. The obvious inaccuracies are as nothing compared with the high-sounding interpretations, which, by the way, are made not by a physician but by one whose name is followed on the letterhead by Ph.D.

This letter is not, I believe, an isolated example of the practice of scientific medicine as it is occasionally done in these days of rackets and high-pressure laboratory tests. Such reports, although not so obviously flagrant, have been known to emanate from other similar laboratories which indulge in a so-called complete examination of a patient. Several questions suggest themselves: Is it advisable for the Massachusetts Medical Society to take cognizance of such fancy quackery since the harm which must inevitably result is probably greater by far than that performed by well-recognized quacks? Secondly, does not this report written by a "Ph.D." and suggesting various therapeutic procedures constitute the practice of medicine without a license?

I would be glad to furnish you with the complete letter if it is requested.

William Dameshek, M.D.
371 Commonwealth Avenue,
Boston, Mass.

## CLOUD APPEARANCES RESEMBLING PATHOLOGIC CONDITIONS AS SHOWN IN X-RAY PICTURES

Editor, *New England Journal of Medicine*,

To lie on one's back on some grassy elevation under a wide expanse of sky and watch the ever-changing cloud effects is surely not one of the lesser joys of living. Cumulus clouds take remarkable shapes. If one has in a long life viewed a good many x-ray films, comparison of these cloud effects and certain x-ray pictures indicating human pathologic processes of various kinds seems inevitable.

Different cloud shapes will "stay put", giving plenty of time for the observer to make his comparisons in each case. One may observe a cloud resembling almost exactly the thick, heavy and irregular tibia of a chronic osteomyelitis — yes, sequestra and cavity formations and all. The thick and more regular and more uniformly sclerosed tibia of specific osteoperiostitis is also simulated to a startling degree. Occasionally one sees the reproduction of a femur, the seat of osteogenic sarcoma, sharp eccentric rays of cloud line simulating the narrow bone production lines radiating outward from the bone tumor mass.

Clouds resembling in shape various long bones with a dark oval area at one end simulating the x-ray appearance of a bone cyst sometimes form under the pleased observer's eye. And then there is the flat fragment of cloud, a little fuzzy around the edges, moth-eaten through its substance, the size and shape of an ilium affected with multiple myeloma. Occasionally a small mass of cumulus shapes up to the size and appearance of the x-ray picture of a stomach, even to a sharp filling defect!

Are these picture resemblances too fanciful?

Let the doubter look up from a similar position as described, to a wide expanse of sky, on some summer day, and see for himself.

WM. PEARCE COUES, M.D.

Brookline, Mass.,
January 29, 1936.

## RECENT DEATH

**SMITH**—GEORGE CARROLL SMITH, M.D., of 369 Tappan Street, Brookline, Mass., whose office was at 483 Beacon Street, Boston, died at his home, February 8, after a short illness.

Dr. Smith was born in West Gardiner, Maine, in 1853, the son of George Lincoln Smith and Abigail Tappan Smith. His early education was acquired at Horseshoe Pond Academy, Monmouth Academy, the Waterville Classical Institute, Bates College and Brown University College. He graduated from the last-named institution in 1876. Before engaging in the study of medicine he taught in several Massachusetts and Connecticut academies.

Dr. Smith received his medical degree from the New York University Medical College in 1881, and subsequently practiced in Natick for twelve years. He later studied for three years in Berlin, Vienna and Paris and on his return made his home in Brookline. He had continued to see patients up to a week before his death.

In addition to articles in medical journals, he was the author of the book What to Eat and Why. Dr. Smith was especially interested in dietetics and chronic diseases.

His memberships included the Massachusetts Medical Society, which he joined in 1882. He was also a Fellow of the American Medical Association and a member of the Boston Medical Library.

His social affiliations were with the University and Boston City Clubs and the Boston Chamber of Commerce.

## OBITUARY

### FRANCIS X. MAHONEY, M.D.

The personnel of the Boston Health Department who have been privileged to serve under Dr. Francis X. Mahoney and to enjoy the warmth of his friendship, many of them for almost a quarter of a century, take this opportunity to pay tribute to his memory and to express their appreciation of his loss to the community.

It is fitting indeed to comment on the duration of his tenure of office. He became a member of the Board of Health in 1910 and, shortly after, was made Chairman of the Board. In 1915, following its reorganization, the value of Dr. Mahoney's services in this field was given recognition by his appointment to the position of Health Commissioner which post he held almost uninterruptedly until the day of his death, with the exception of one term.

Dr. Mahoney's services as Commissioner are, of course, closely related to the improvement of the public health in Boston. When he assumed the duties and responsibilities of Commissioner of Health, the infant mortality rate of our city was 111 deaths annually for every thousand babies born. Constant improvement in this very sensitive field of health service reduced the rate to approximately fifty, thus resulting in an annual saving of hundreds of lives of our babies.

When Dr. Mahoney first became Health Commissioner, almost 1500 died annually in our city from tuberculosis. This number has been cut to nearly one-third. Up to a few years ago, close to 200 children died annually from diphtheria. Last year this figure dropped to ten. Typhoid fever, which caused over fifty deaths each year, has been reduced to a minimum as a cause of death.

Many other facts could be cited as an indication of the improvement of the public health in Boston in recent years.

Dr. Mahoney's friends have too kind a memory of his modesty to claim for him the sole credit for this betterment in the health conditions in our city. Moreover, they have a fond recollection of not only his interests in the health of Boston's residents, but also his earnest endorsement of every possible effort for the conservation of life and the prevention of disease.

Above all, they recognize the latitude given to his subordinates in the direction of the individual departments so that the program as a whole might be well organized, and more efficient health work carried on.

He supported strongly the plan for the expansion of the George Robert White Fund Health Units and the development of the Child Hygiene Program of the City. On every possible occasion, he vigorously advocated the need for greater participation by the

rank and file of the medical profession in the practice of preventive medicine. He was mindful of the significance and importance of a clean water and milk supply and did everything to encourage high standards in these vital essentials to everyday life. He championed strongly the extension of the Tuberculosis Clinics on a city-wide basis, so that there might be made available facilities for the very early recognition of the disease.

His genial nature, his warm smile, and his fine character endeared him to us all, and we join with his army of friends in expressing deep sympathy to his family and grateful appreciation of his unselfish services to the City of Boston.

## NOTICES

### ANNOUNCEMENT OF THE FRANCIS AMORY SEPTENNIAL PRIZE OF THE AMERICAN ACADEMY OF ARTS AND SCIENCES UNDER THE WILL OF FRANCIS AMORY

In compliance with the requirements of a gift under the will of the late Francis Amory of Beverly, Massachusetts, the American Academy of Arts and Sciences announces the offer of a septennial prize for outstanding work with reference to the alleviation or cure of diseases affecting the human genital organs, to be known as the Francis Amory Septennial Prize. The gift provides a fund, the income of which may be awarded for conspicuously meritorious contributions to the field of knowledge "during the said septennial period preceding any award thereof, through experiment, study or otherwise . . . in the diseases of the human sexual generative organs in general." The prize may be awarded to any individual or individuals for work of "extraordinary or exceptional merit" in this field.

In case there is work of a quality to warrant it, the first award will be made in 1940. The total amount of the award will exceed ten thousand dollars, and may be given in one or more awards. It rests solely within the discretion of the Academy whether an award shall be made at the end of any given seven-year period, and also whether on any occasion the prize shall be awarded to more than a single individual.

While there will be no formal nominations, and no formal essays or treatises will be required, the Committee invites suggestions, which should be made to the Amory Fund Committee, care of the American Academy of Arts and Sciences, 28 Newbury Street, Boston, Massachusetts, U. S. A.

### THE JOURNAL CLUB OF THE DEPARTMENT OF OBSTETRICS, HARVARD MEDICAL SCHOOL

The monthly meeting of the Journal Club of the Department of Obstetrics, Harvard Medical School, will be held on February 20, 1936, at 8:15 P.M. at the Boston Lying-in Hospital.

Dr. Judson Smith will review papers on the treatment of habitual and threatened miscarriages together with recent work on the physiology of corpus luteum. The pathological aspects of miscarriages and abortions will also be presented.

Professor Hisaw of the Harvard University will discuss the work on the physiology of the corpus luteum.

ARTHUR T. HERTIG, M.D., *Chairman.*

### MEDICAL CLINIC AND STAFF ROUNDS AT THE PETER BENT BRIGHAM HOSPITAL

At 3:30 P.M. on Thursday, February 20, in the Amphitheatre of the Peter Bent Brigham Hospital, Dr. Henry A. Christian, Physician-in-Chief, Hersey Professor of the Theory and Practice of Physic in the Harvard Medical School, will give a medical clinic. To it are cordially invited practitioners and medical students.

### REMOVAL

WARREN D. RUSTON, M.D., announces the removal of his office to 29 Commonwealth Avenue, Boston.

## NOTICES OF MEETINGS

### HARVARD MEDICAL SOCIETY

The next meeting of the Harvard Medical Society will be held in the Peter Bent Brigham Hospital Amphitheatre (Shattuck Street Entrance), Tuesday evening, February 25, at 8:15 P.M.

PROGRAM

Presentation of Cases.

Spontaneous Hypoglycemia. By Dr. Russell M. Wilder, Mayo Clinic, Rochester, Minnesota.

Medical students and physicians are cordially invited to attend.

MARSHALL N. FULTON, M.D., *Secretary.*

### NEW ENGLAND HEART ASSOCIATION

The next meeting of the New England Heart Association will be held at the Memorial Hospital, Worcester, Mass. (Knowles Hall, Nurses' Home), Monday, February 24, 1936, at 8:00 P.M.

PROGRAM

1. Effects of Contagious and Infectious Diseases on the Heart.
   - A. General Statement of Main Topic. Dr. O. H. Stansfield.
   - B. Late Results of Contagious and Infectious Diseases on the Heart. Dr. E. H. Halloran.
2. Acute Benign Pericarditis. Dr. F. B. Carr.
3. Coronary Symptoms in Pernicious Anemia. Dr. J. J. Dumphy.

All members of the New England Heart Association and interested physicians are invited to attend.

JAMES M. FAULKNER, M.D., *Secretary.*

### NEW ENGLAND OPHTHALMOLOGICAL SOCIETY

The 308th meeting of the New England Ophthalmological Society will be held on Tuesday, February 18, at the Massachusetts Eye and Ear Infirmary, 243 Charles Street, Boston.

9:00 A.M.—Clinic and Operating Room.
11:30 A.M.—Neuro-Ophthalmological Conference. Applicant for Membership.

8:00 P.M.

Simple Technique for Plotting Diplopia. Dr. William D. Rowland.

Paper:

History of Ophthalmology as a Specialty in New England. Dr. Allen Greenwood.

### MASSACHUSETTS MEMORIAL HOSPITALS

There will be a meeting of the Surgical Section in the Ladies' Aid Room, Talbot Memorial, 82 East Concord Street, Boston, on Friday, February 14, at 12 noon.

Papers will be presented by Dr. Charles Sziklas and Dr. David B. Stearns.

MILO C. GREEN, M.D., *Secretary.*

### NEW ENGLAND PHYSICAL THERAPY SOCIETY

The regular meeting of the New England Physical Therapy Society will be held at the Hotel Kenmore, Boston, on Wednesday evening, February 19, at 8 o'clock.

PROGRAM

Elementary Physics of Galvanism. L. L. Campbell, Ph.D., Professor of Physics, Emeritus, Simmons College.

Clinical Uses of the Galvanic Current:

Iontophoresis. Gynecological Conditions. Frederick H. Morse, M.D. Pioneer in Electrotherapy.

Nerve Conditions. H. Houston Merritt, M.D., Associate in Neurology, Harvard Medical School.

Some Common Conditions in General Practice. Claude L. Payzant, M.D., Chief, Department of Physical Therapy, Quincy Hospital.

The Council will meet at 6 P.M.

Members and guests will meet for dinner at 6:30 in the Empire Room at the Kenmore.

All members of the medical profession are cordially invited to attend.

WILLIAM D. MCFEE, M.D., *Secretary.*

41 Bay State Road, Boston.

### BOSTON MEDICAL HISTORY CLUB

8 Fenway, Boston

Monday, February 17, at 8:15 P.M., at the Boston Medical Library.

Domenico Cotugno—His Description of the Cerebrospinal Fluid. Henry R. Viets, M.D.

A Belated Eulogy to John H. Watson, M.D. Reginald Fitz, M.D.

BENJAMIN SPECTOR, M.D., *Secretary.*

### A JOINT MEETING TO DISCUSS A COMMUNITY PLAN FOR MEDICAL CARE

The Boston Council of Social Agencies, the Boston Health League and the Hospital Council of Boston have issued invitations to a meeting in Sprague Hall, Boston Medical Library, 8 Fenway, on Monday, February 17, at four o'clock.

Ross Garrett, Co-Ordinator for the Medical Economic Security Council in operation in Washington, D. C., will explain the plan which includes hospital insurance; maintains a Service Bureau to assist in the payment of professional fees; attempts to determine the ability of the patient to pay, and regulates the distribution of the burden of free care among all hospitals.

Since this plan, which is sponsored by the Council of Social Agencies in Washington, is being watched with great interest in other parts of the country, we are fortunate in having this opportunity of discussing it with Mr. Garrett. It is hoped that staff and board members of organizations interested in this subject will be present.

Mr. Garrett's visit is made possible through the coöperation of the Massachusetts Medical Society, the Massachusetts Dental Society and the Hospital Council of Boston.

*This Problem Concerns Everyone.*

### THE NEW YORK HARVEY SOCIETY

The fifth lecture of the Harvey Society will be given at the New York Academy of Medicine on February 20 by Dr. John F. Fulton, Sterling Professor of Physiology, Yale University School of Medicine, on The Interrelation of Cerebrum and Cerebellum in the Regulation of Somatic and Automatic Functions.—*Science.*

## SOCIETY MEETINGS, CONGRESSES AND CONFERENCES

### CALENDAR OF BOSTON DISTRICT FOR THE WEEK BEGINNING MONDAY, FEBRUARY 17, 1936

**Monday, February 17—**

*4 P. M. Joint Meeting to Discuss a Community Plan for Medical Care. Boston Medical Library, 8 Fenway, Boston.

8:15 P.M. Boston Medical History Club at the Boston Medical Library, 8 Fenway, Boston.

**Tuesday, February 18—**

*9-10 A.M. Boston Dispensary, 25 Bennet Street, Boston. X-Ray Demonstration. Dr. Alice Ettinger.

9 A.M., 11:30 A.M. and 8 P.M. New England Ophthalmological Society. Massachusetts Eye and Ear Infirmary, 243 Charles Street, Boston.

*12 M. South End Medical Club. Office of the Boston Tuberculosis Association, 554 Columbus Avenue, Boston.

2:30 P.M. Pediatric Ward Visit. Massachusetts Eye and Ear Infirmary.

**Wednesday, February 19—**

*9-10 A.M. Boston Dispensary, 25 Bennet Street, Boston. Auscultation of the Abdomen. Dr. Neil Stevens.

†12 M. Clinico-Pathological Conference. Children's Hospital.

8 P.M. New England Physical Therapy Society. Hotel Kenmore, Boston.

**Thursday, February 20—**

*8:30-9:30 A.M. Clinic, Surgical Staff of the Peter Bent Brigham Hospital, at the Peter Bent Brigham Hospital.

*9-10 A.M. Boston Dispensary, 25 Bennet Street, Boston. Heart Clinic. Dr. Samuel H. Proger.

*3:30 P.M. Medical Clinic at the Peter Bent Brigham Hospital.

**Friday, February 21—**

*9-10 A.M. Boston Dispensary, 25 Bennet Street, Boston. Some Aspects of Clinical Endocrinology. (With Motion Pictures.) Dr. Lewis M. Hurxthal.

**Sunday, February 23—**

4 P.M. Free Public Lecture, Harvard Medical School, Building D, Longwood Avenue. The Rôle of the White Blood Cells in Health and Disease. Dr. Henry Jackson, Jr.

*Open to the medical profession.
†Open to Fellows of the Massachusetts Medical Society.

**February 14**—William Harvey Society. Auditorium, Beth Israel Hospital, Boston, at 8 P.M.

**February 14**—Massachusetts Memorial Hospitals, Surgical Section. See page 339.

**February 17**—A Joint Meeting to Discuss a Community Plan for Medical Care. See page 339.

**February 17**—Boston Medical History Club. See page 339.

**February 18**—The South End Medical Club. Office of the Boston Tuberculosis Association, 554 Columbus Avenue, Boston, at 12 noon.

**February 18**—New England Ophthalmological Society. See page 338.

**February 19**—New England Physical Therapy Society. See page 339.

**February 20**—Medical Clinic, Peter Bent Brigham Hospital. See page 338.

**February 20**—New York Harvey Society. See page 339.

**February 20**—The Journal Club of the Department of Obstetrics, Harvard Medical School. See page 338.

**February 24**—New England Heart Association. See page 338.

**February 24**—Springfield Medical Association, 8:30 P.M. at the rooms of the Springfield Academy of Medicine, 20 Maple Street.

**February 24 to May 16**—International Medical Postgraduate Courses in Berlin. See page 1211, issue of December 12, 1935.

**February 25**—Harvard Medical Society. See page 338.

**March 2-6**—The American College of Physicians. See page 91, issue of January 9.

**April 20-24**—A Postgraduate Institute in Philadelphia. See page 224, issue of January 30.

**June 15-19**—The Executive Board of the Catholic Hospital Association will meet at the Fifth Regiment Armory, Baltimore, Md.

**September, 1936**—First International Conference on Fever Therapy. See page 1325, issue of December 26, 1935.

**October 19-23**—Clinical Congress of the American College of Surgeons. See page 180, issue of January 23.

## DISTRICT MEDICAL SOCIETIES

### ESSEX SOUTH DISTRICT MEDICAL SOCIETY

**March 4**—Wednesday. Lynn Hospital. Clinic 5 P.M. Dinner 7 P.M. Speaker: Dr. Timothy Leary. Subject: Arteriosclerosis.

**April 1**—Wednesday. Essex Sanatorium, Middleton. Clinic 5 P.M. Dinner 7 P.M. Speaker: Dr. Richard H. Overholt of the Lahey Clinic. Subject: Chest Surgery.

**May 7**—Thursday. Censors' Meeting.

**May 13**—Wednesday. Annual Meeting. Salem Country Club. Dinner at 7 P.M. Speaker: Dr. Paul White. Subject to be announced later.

R. E. STONE, M.D., Secretary.
88 Lothrop Boulevard, Beverly.

### FRANKLIN DISTRICT MEDICAL SOCIETY

Meetings are held on the second Tuesdays of March and May at the Weldon Hotel, Greenfield, at 11 A.M.

CHARLES MOLINE, M.D., Secretary.
Sunderland.

### MIDDLESEX EAST DISTRICT MEDICAL SOCIETY

Meetings to be held at the Bear Hill Golf Club, Stoneham, at 12:15 P.M.

**March 11, May 6.**

K. L. MACLACHLAN, M.D., Secretary.
1 Bellevue Avenue, Melrose.

### NORFOLK DISTRICT MEDICAL SOCIETY

**February 25**—Massachusetts Memorial Hospitals at 8 P.M. Papers by the staff.

**March 31**—Hotel Kenmore, at 8 P.M. Dr. Benedict F. Boland—Cauterization of the Cervix Uteri Using Various Electrical Methods. Illustrated with lantern slides.

**May**—Annual Meeting. (Place, date and subject to be announced.)

The censors meet for the examination of candidates: May 7, 1936; November 5, 1936.

FRANK S. CRUICKSHANK, M.D., Secretary.
1236 Beacon Street, Brookline.

### PLYMOUTH DISTRICT MEDICAL SOCIETY

**March 19**—Plymouth County Sanatorium, South Hanson.

**April 16**—Brockton Hospital.

**May 21**—Lakeville State Sanatorium.

G. A. MOORE, M.D., Secretary.
167 Newbury Street, Brockton.

### SUFFOLK DISTRICT MEDICAL SOCIETY

**March 18**—Meeting at the Boston Medical Library. The Laboratory and Clinical Story of Fatigue, Dr. Arlie V. Bock and Dr. David B. Dill. Discussion: Dr. Donald J. MacPherson and Dr. Augustus Thorndike, Jr.

**April 29**—Annual Meeting at the Boston Medical Library. The Treatment of Septicaemia, Dr. Champ Lyons. The Pleurality of Scarlatinal Streptococcus Toxin, Dr. Sanford B. Hooker. Discussion: Dr. Hans Zinsser.

The medical profession is cordially invited to attend all of these meetings.

ROBERT L. DeNORMANDIE, M.D., President,
CHARLES C. LUND, M.D., Secretary.

### WORCESTER DISTRICT MEDICAL SOCIETY

**March 11**—Wednesday evening. Memorial Hospital, Worcester, Mass. Dinner and scientific program. Subjects of program to be announced later.

**April 8**—Wednesday evening. Hahnemann Hospital, Worcester, Mass. Dinner and scientific program. Subjects of program to be announced later.

**May 13**—Wednesday afternoon and evening. Annual Meeting of Society. Time, place and details of program to be announced in an April issue of the Journal.

ERWIN C. MILLER, M.D., Secretary.
27 Elm Street, Worcester.

## BOOK REVIEW

**The Sexual Relations of Mankind.** Paolo Mantegazza. 335 pp. New York: Eugenics Publishing Company. $6.00.

Singularly modern in its point of view is this translation of Mantegazza's Gli amori degli uomini, first published in 1885. Mantegazza was a physician, laboratory-worker, traveler and anthropologist; this compendium of the love-customs of the tribes and races of the world reflects the mental attributes of a man educated as he was. At the same time he was an Italian, and as he himself tells us (p. 290) "As I see it (and I trust no one will take offense at this), the Italians are the foremost lovers among civilized races."

The book consists of a great collection of facts regarding various phases of the sexual side of life, including the Festivals of Puberty, The Sexual Embrace and Its Forms, Mutilation of the Genitals, Sexual Choice, Position of the Woman in Marriage, Monogamy, Polygamy and Polyandry and so forth. The customs of many outlandish tribes, some of whom the author himself had visited, are presented in a scientific manner, free from sensationalism. It has become one of the source-books for this sort of information. The presentation of these facts is often accompanied by Mantegazza's comment; at the end of the book he submits a list of what he considers to be the first signs of a better future for love. These points, written fifty years ago, might well be taken from the present-day writings of one of our most free-thinking writers. They are as follows:

> "Less ignorance of sexual matters on the part of young girls.
>
> "Free choice on the part of both sexes, in place of a contract imposed by parents and endured by their offspring.
>
> "Less hypocrisy.
>
> "Restitution of its dignity to marriage, divorce being surrounded by wise precautions.
>
> "And finally—and do not be scandalized by this—a sincere and clean-cut separation of free and sexual love from that troth which is plighted between two creatures, who have come to know each other thoroughly well over a period of time, and who are animated by a desire to found a family."